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BRITISH MEDICAL JOURNAL

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ADRENALECTOMY FOR BREAST CANCER*

BY

Sir STANFORD CADE, K.B.E., C.B., F.R.C.S., F.R.C.O.G., M.R.C.P.

Senior Surgeon, Westminster Hospital

It is now recognized that cancer of the breast and prostate are hormone-dependent tumours—that is, cancers which can be controlled by the administration of sex hormones. That removal of the ovaries influences advanced breast cancer has been known since 1896, when George Beatson published the first reports on this method of treatment. By 1905 Hugh Lett had reported 99 cases of oophorectomy for the late stages of mammary carcinoma. The popularity of this operation waxed and waned during the following half-century owing to the development of radiation-induced menopause. To-day it is thought by many that radiation menopause is not equivalent to surgical castration and that it does not abolish oestrogen formation. Huggins and Bergenstal (1951, 1952) demonstrated that the adrenal cortical hormones sustain mammary cancer and that their withdrawal is followed in a number of cases by marked regression of the tumour. Prolonged survival of adrenalectomized patients on cortisone maintenance has given a great impetus to this method of treatment (West *et al.*, 1952; Taylor *et al.*, 1953; Pyrah and Smiddy, 1954).

Huggins has drawn attention to the following important observations: (1) Gonadectomy is followed by a compensatory hypertrophy of the adrenals and by increased cortical function. (2) In men, removal of the testis is followed by an increase of 17-ketosteroids in the urine, and these high values are eliminated by bilateral adrenalectomy. (3) Following oophorectomy, women continue to excrete oestrogens in the urine, but these compounds disappear from the urine after adrenalectomy. Further, Huggins and Dao (1953) formed the opinion that in a number of post-menopausal women the ovaries continue to have an active endocrine function, which indicates oophorectomy even after the menopause.

The rationale of bilateral oophorectomy and adrenalectomy is therefore the elimination of all known sources of hormones which can influence the growth of mammary cancer and the creation of a physiological environment poor in steroids.

The Natural Course of Breast Cancer

Mammary cancer varies in its rate of growth and power of dissemination from the slow and apparently harmless course of the "atrophic scirrhus" on the one hand to the rapidly fatal course of the "acute inflammatory" type on the other. The variation noted in different histological types, in different age groups, in different hormonal states (pre- and post-menopausal and in pregnancy and lactation) indicates that the hormonal influence is variable and that some tumours are hormone-dependent to a greater degree than others and that some are independent of hormones. But in nearly all cases after a variable period of quiescence and localization the disease spreads locally, to the regional lymph nodes, to the skeleton, and to the viscera.

The early and localized stage of the disease can be controlled by surgical treatment; moderate extension can be alleviated by radiotherapy; in the early stages of dissemination the use of androgens or oestrogens may arrest the spread of the disease for variable periods. Bilateral adrenalectomy is indicated when the disease is disseminated and no longer amenable to treatment by surgical, radiological, and hormonal means. It is therefore a most important advance in the treatment of cancer of the breast and should be considered in patients in whom the disease is no longer amenable to the established methods of treatment.

This report is based on the experience of 69 bilateral adrenalectomies, 56 of these in advanced and disseminated breast cancer. The study of these patients has shown that about one-third are no longer under hormonal influence, and in these no benefit is obtained; that in the remaining two-thirds there is an improvement varying from complete control of the disease to an amelioration which varies in degree and in the length of time which the improvement lasts. The longest period of improvement in this series is 24 months. No other method of treatment has as yet achieved control of disseminated, widespread, and often terminal stages of breast cancer comparable to that following bilateral adrenalectomy.

Selection of Cases

In all the 56 adrenalectomized patients cancer of one or both breasts had been present for various periods of time and had been treated previously according to its stage and extent by appropriate methods. In every patient skeletal, visceral, or soft-tissue metastases were widespread and the expectation of life was very limited. The selection of patients was, in fact, simple: only those to whom nothing further could be offered were submitted to adrenalectomy. The operation was therefore put to a severe test and was deemed a justifiable experiment. It was soon found that patients with disseminated disease in the pulmonary lymphatics, with involvement of the pericardium and the heart, were not likely to benefit from adrenalectomy and that the

*Based on a Hunterian Lecture given at the Royal College of Surgeons of England on June 17, 1954.

operative risk in such cases was high. In fact, involvement of the lungs and heart to an extent which impaired vital function is regarded at present as the main contraindication for the operation.

Management

Pre-operative.—All the patients are carefully assessed, as regards the extent of the disease and the general state of health, by clinical, radiological, and chemical investigations. In addition the output of 17-ketosteroids and oestrogens is studied and the Thorn test for adreno-cortical function done. The pre-operative preparation consists in the correction of the blood state by transfusion and the administration of cortisone. In this series of patients cortisone only was used. There seemed to be no need for D.C.A. (deoxycortone) or for extra salt. Cortisone acetate is given in 100-mg. doses by intramuscular injection forty-eight, twenty-four, and one hour before the operation.

Post-operative.—During the operation and in the immediate post-operative period noradrenaline is used only if there is a marked fall in the blood pressure. Noradrenaline is not used as a routine, only as an emergency measure. On the first post-operative day cortisone is given by injection in 100-mg. doses six hourly; this is reduced by a half on the second post-operative day; from the third to the fifth post-operative day 25 mg. of cortisone acetate is given by mouth six-hourly; this is further reduced to 75 mg. a day and finally to 50 mg. daily as a maintenance dose. Patients are given instructions about the need of extra cortisone during intercurrent illness or following an operation or an accident. Medullary failure occurs very rarely, sometimes several weeks or even months after adrenalectomy, and is corrected by noradrenaline.

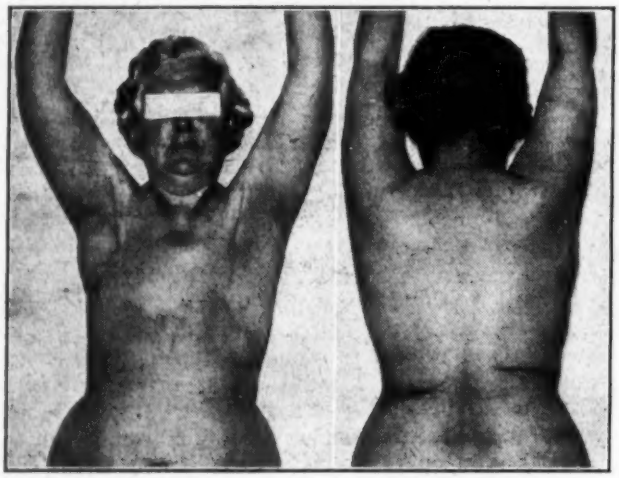

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FIG. 1.—Case 1. Bilateral carcinoma of breast. Multiple skeletal metastases. Survival to date: 24 months after adrenalectomy. Marked increase in weight; regression of skeletal lesions. A, Moon-shaped face. B, Scars of bilateral adrenalectomy.

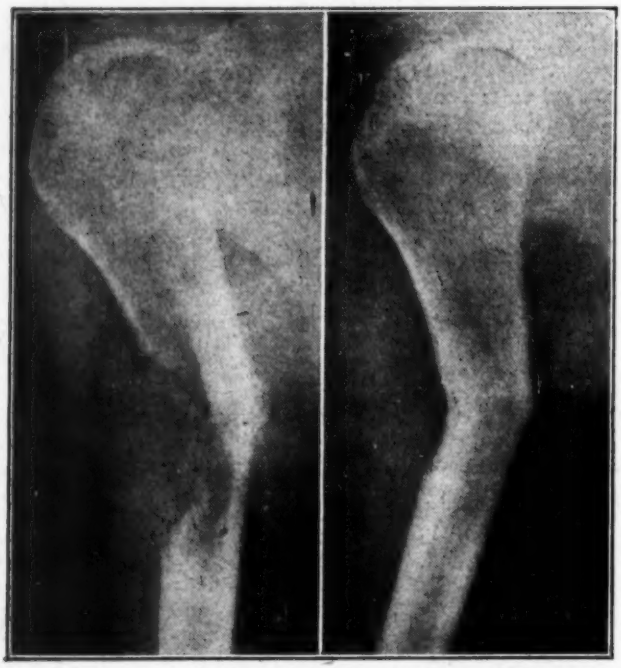

FIG. 2.—Case 2. Metastasis in humerus before and 10 months after adrenalectomy.

The Operation

Bilateral adrenalectomy and oophorectomy can be done in one stage. The practice at present is to carry out bilateral oophorectomy and unilateral adrenalectomy in one stage and postpone the second adrenalectomy for seven days. The best approach to the adrenal is through the bed of the twelfth rib, the patient lying on her side, in the position usually adopted for renal operations. When the twelfth rib is small, it is necessary to remove the eleventh rib. The operation is technically simple, but the adrenal must be handled with care as it is friable, and in some patients there occurs a marked fall or rise in the blood pressure when the gland is manipulated. Haemorrhage is controlled by careful ligation of the adrenal veins, which show considerable anatomical variations, and with which the surgeon should be familiar. Accidents which can be guarded against are injury to the pleura, the renal vein, or inferior vena cava. In most patients convalescence is smooth, and the post-operative course gives rise to anxiety only in patients with cardiac or pulmonary involvement by disease or following operative difficulties.

Results

Mortality.—Of the 56 patients, 8 died following the operation, a mortality of 13.6%. The causes of death in these cases were: one from pulmonary embolus; one from cardiac arrest during the operation; two from renal failure on the sixth and twenty-first post-operative days; one from cerebral metastasis; and three from pleural and pericardial involvement by disease. A most careful study of these cases indicates that the very precarious state of the patient as well as the operative risk accounted for the deaths. It is interesting to note that there has been no operative death in the last 27 cases.

Failures.—Twelve patients derived no benefit at all from the operation: the disease progressed, and most patients died from it within a period of a few months. In 14 patients there was no alteration in the metastases, although some improvement in the general state of health and relief of symptoms was obtained. In four patients there was no relief of symptoms, although local improvement of the lesions was noted.

Successful Cases.—These patients have been studied as regards subjective and objective improvement. In 13 patients the results were remarkable, in the sense that bedridden patients with severe pain became ambulatory and no longer required pain-relieving remedies. The subjective improvement was accompanied by increase of weight and a return to a near-normal life. Objective improvement was assessed by healing of pathological fractures, alteration of osteolytic lesions to a normal bone structure, shrinkage of visible external tumours, such as the primary tumour, skin nodules, enlarged lymph nodes, and disappearance of metastases in the omentum, peritoneum, lungs, liver, and in the choroid. In one patient, nearly *in extremis* when adrenalectomized, with extensive destruction of the base of the skull, multiple cranial nerve palsies, and pleural, pulmonary, and intra-abdominal metastases, most lesions disappeared and the patient regained a near-normal state of health for a period of fourteen months, when the disease became active again

and the patient died fifteen months after adrenalectomy. So far, the longest survival period in this series is twenty-four months and the shortest six weeks. Examples reported here show regression of various types of lesions—skeletal, visceral, and soft tissue. Improvement can usually be noticed within a few days of the operation. If no improvement occurs within one week the case is likely to prove a failure.

Prognosis.—There is no method at present by which to forecast if adrenalectomy will be a success or a failure. Attempts have been made to correlate the results with the age of the patient, the extent of the disease, and the histological type of the primary growth. Both successes and failures were noted in all histological types, although Huggins and others think that the adenocarcinoma is more likely to regress than the carcinoma simplex or other varieties. The incidence of metastases in the adrenal is very high and reproduces accurately the histological variety of the primary growth. The Table shows the results in 48 patients.

Results in 48 *Patients: Adrenalectomy for Breast Cancer*

	Alive		Dead
	Subjective	Objective	
Excellent	13	10	1 (14 months)
Satisfactory	22	16	1 (12 months)
No effect	7	14	9
Too early for assessment..	6	8	—
	48	48	11

Illustrative Cases

The following successful cases illustrate the benefit which has been achieved by adrenalectomy in some patients with advanced and disseminated breast cancer no longer treatable by other methods. The cases chosen show the effect on skeletal, pulmonary, and soft-tissue metastases.

Case 1. Aged 50. Right radical mastectomy in January, 1950, for a carcinoma simplex with involvement of axillary lymph nodes. Post-operative *x*-ray therapy. In August, 1951, left radical mastectomy for a histologically similar tumour; lymph nodes in axilla involved. Post-operative *x*-ray therapy. In January, 1952, multiple skeletal metastases in spine, ribs, and skull. Testosterone propionate given by injection. In July, 1952, bilateral oophorectomy; both ovaries infiltrated by metastases. In November, 1952, the skeletal metastases had spread widely. Nearly all the ribs and all the vertebrae were involved. The sternum was the seat of a large tumour; 17-ketosteroid excretion: 6 to 7 mg./24 hours. Bilateral adrenalectomy performed. This was followed by immediate relief of pain and a fall in the 17-ketosteroid excretion to 2.5 mg./24 hours, and the Thorn test showed no fall in the eosinophil count. Both subjective and objective improvement was very marked. The effect on the skeletal metastases in the skull, sternum, and spine was a return of the bones to a normal structure. The patient remains well to date, twenty-four months after adrenalectomy. Fig. 1 illustrates the patient's present appearance; there is an alteration in her face (moon-shaped) due to the cortisone; marked increase in weight and complete relief from pain.

Case 2.—Aged 42. Right radical mastectomy for a pleomorphic carcinoma of the breast in December, 1950. Post-operative *x*-ray therapy. In April, 1952, skeletal metastasis in lumbar and dorsal vertebrae. Pain relieved by *x*-ray therapy, but further metastases in the spine developed. From July to October, 1952, given testosterone propionate, but extensive skeletal metastases found in right humerus (pathological fracture), skull, ribs, pelvis, femora; collapse of third lumbar vertebra. Patient was bedridden and in considerable pain on movement. 17-ketosteroids, 9.4 mg./24 hours. Bilateral adrenalectomy performed on April 9, 1953. Both adrenals contained metastases. Skeletal pain relieved within twenty-four hours. Rapid recovery; healing of pathological fracture of humerus and other skeletal metastases. Fig. 2 shows the right humerus before adrenalectomy and ten months later; Fig. 3 shows extensive osteolytic lesions of the skull before and five months after operation. The patient is ambulatory and remained well seventeen months after adrenalectomy.

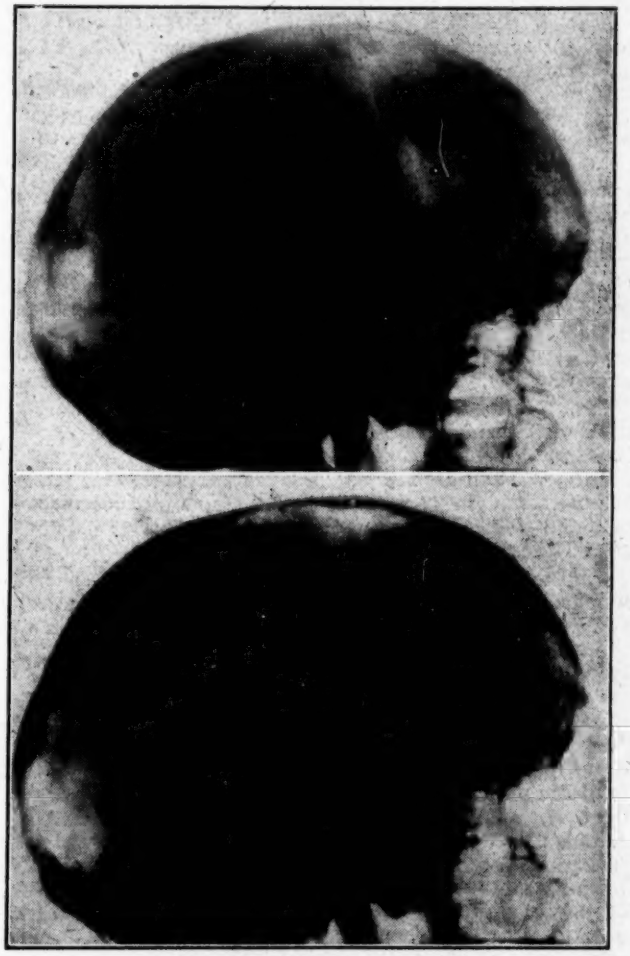

FIG. 3.—Case 2. Osteolytic lesions in right parietal and frontal bones. Before and five months after adrenalectomy.

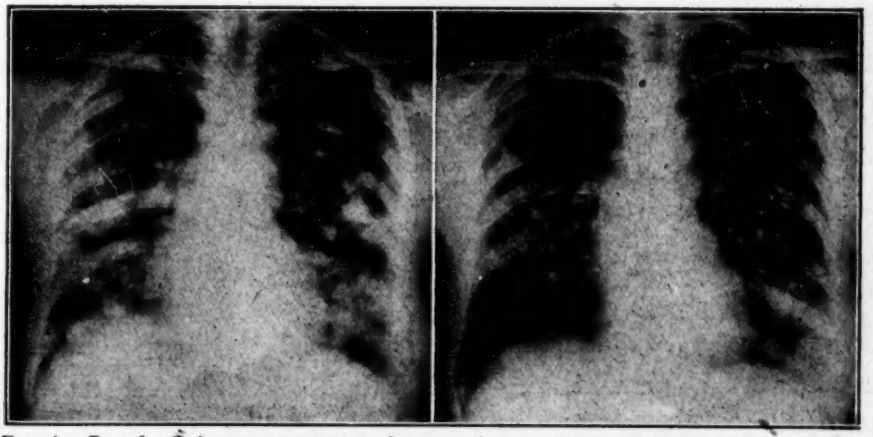

FIG. 4.—Case 3. Pulmonary metastases from carcinoma of breast before and three months after adrenalectomy.

Case 3.—Aged 53. In September, 1952, mastectomy and post-operative *x*-ray therapy for an adenocarcinoma of the right breast. In August, 1953, patient noticed "flashes of light" in the right eye with progressive visual impairment. Seen by Mr. E. F. King at Moorfields Hospital and found to have a large choroidal metastasis in the right eye. *X*-ray examination of chest showed extensive bilateral pulmonary metastases (Fig. 4). Bilateral oophorectomy and adrenalectomy in October, 1953; the right ovary contained a metastasis; no metastases in either adrenal. Rapid improvement followed; complete disappearance of choroidal metastasis within three weeks and nearly complete regression of pulmonary metastases within three months (Fig. 4). Patient remains well to date, twelve months after adrenalectomy.

Case 4.—Aged 35. In November, 1951, radical mastectomy for an adenocarcinoma of the right breast with metastases in the axillary lymph nodes. Post-operative *x*-ray therapy. In July, 1952, metastases in the ribs and dorsal spine with destruction of several mid-dorsal vertebrae and marked kyphosis. *X*-ray menopause and testosterone failed to control the disease. In November, 1953, there was widespread involvement of the skull, pelvis, lower dorsal and lumbar vertebrae, ribs, both scapulae, and both femora. Sternal puncture showed numerous malignant cells in the bone marrow. Bilateral adrenalectomy performed on November 10. Rapid recovery; complete relief of bone pain. Increase in weight by 2 stone (12.7 kg.) and return to duty as a nurse in an administrative capacity. Improvement lasted nine months. Died twelve months after adrenalectomy.

Case 5.—Aged 43. Left radical mastectomy for a carcinoma simplex of the left breast in March, 1950. Two years later, local recurrence in scar (Fig. 5,A) which was not controlled by radiotherapy. Bilateral oophorectomy and adrenalectomy in November, 1953. Rapid disappearance of lesion (Fig. 5,B). Remains well to date, twelve months after adrenalectomy.

Case 6.—Aged 39. Radical mastectomy for a carcinoma simplex of the right breast in January, 1951. Post-operative *x*-ray therapy. In May, 1952, carcinoma of the left breast which did not respond to *x*-ray therapy. In December, 1952, there was a condition of carcinoma-en-cuirasse on the left side of the chest wall; enlarged supraclavicular lymph nodes. No improvement from testosterone. In February, 1953, bilateral oophorectomy was done with temporary improvement. In October, 1953, there were extensive local disease; an orbital metastasis causing proptosis of the right eye; skeletal metastases in the pelvis and the right scapula; and a pleural effusion. Bilateral adrenalectomy was performed on November 24, 1953. This was followed by remarkable subjective and objective improvement: relief of pain, return to a normal life, and complete disappearance of the lesion on the right side of the chest wall and supraclavicular gland. Remains well to date, twelve months after adrenalectomy.

Case 7.—Aged 40. Right radical mastectomy for a late stage II scirrhous carcinoma of the right breast in October, 1949. Post-operative *x*-ray therapy. Three years and three months later, in January, 1954, metastases in pelvis, femora, and opposite axilla (Fig. 6,A). Bilateral oophorectomy and adrenalectomy in February, 1954. Both adrenals but not the ovaries involved by metastases. Uneventful recovery. Rapid disappearance of contralateral lymph node (Fig. 6,B): complete relief of pain from skeletal metastases to date, eight months after adrenalectomy.

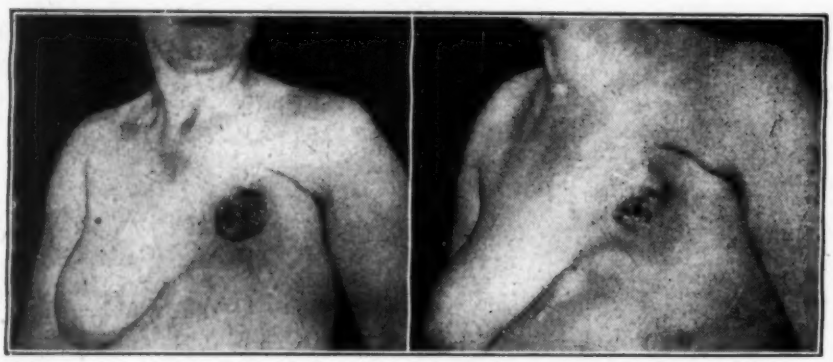

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FIG. 5.—Case 5. Local recurrence following radical mastectomy and radiotherapy. A, Before adrenalectomy. B, Five months after adrenalectomy.

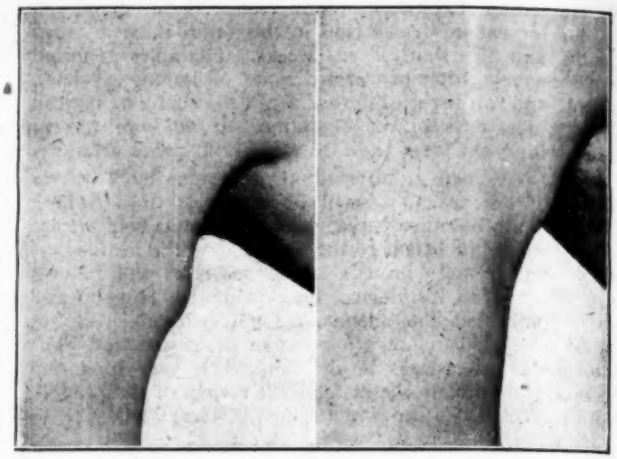

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FIG. 6.—Case 7. Enlarged lymph nodes in contralateral (left) axilla following right radical mastectomy. This patient also had multiple skeletal metastases. A, Before adrenalectomy. B, Five months after adrenalectomy.

Summary and Conclusions

A series of 56 patients is reported on whom bilateral adrenalectomy was performed for disseminated breast cancer. Extended survival on cortisone maintenance has been obtained, so far up to 24 months. In a proportion of cases both subjective and objective improvement has been achieved which has never been accomplished before by any other method of treatment. Relief of pain from skeletal metastases, regression of visible and palpable lesions, reossification of skeletal metastases, and union of pathological fractures have followed adrenalectomy. There is no method, so far, to forecast if a patient will be benefited by the operation or not. Beneficial effects have been achieved in about 60% of patients; and in a proportion of these—23% of all cases—the improvement was quite remarkable and surprising.

The risk of the operation is reasonable and maintenance of life on cortisone simple. Although there is no likelihood of any permanent effect, the results are encouraging, and it seems justifiable to advise the operation at an earlier stage of the disease.

I would like to express my thanks to the Medical Research Council for the provision of cortisone and to my colleagues at Westminster Hospital for their help and co-operation, especially to Dr. F. D. Hart and Dr. C. J. M. Clark for the medical care of the patients, to Mr. A. Jessiman and Mr. G. Westbury for the help at the operations and post-operative care, and to Miss Worcester, the sister in charge of my wards, and her staff, on whom fell the burden of nursing care of these very ill patients.

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PROBLEMS INVOLVED IN PREVENTION OF BACTERIAL COMPLICATIONS OF MEASLES

WITH SPECIAL REFERENCE TO PROPHYLACTIC SULPHADIAZINE*

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There is general agreement that sulphonamides and penicillin have contributed greatly to the fall in the mortality of measles in recent years (*British Medical Journal*, 1952), but the need for further study of the prevention of morbidity arising as a result of measles is also recognized (*British Medical Journal*, 1953). Since this essay was submitted, the *Lancet* (1954), in discussing the prophylaxis of measles complications, makes the following statement: "In this country quite satisfactory prevention of bacterial complications can still be obtained by the simpler and cheaper method of sulphonamide prophylaxis. This is one of the few forms—if not the only form—of routine sulphonamide prophylaxis that can be generally recommended." Two questions, however, have not yet been satisfactorily answered. The first is whether sulphonamides are, in fact, effective in the prophylactic as opposed to the curative treatment of the bacterial complications of measles. The second is whether the preventive dose, if effective, is sufficiently lower than the curative to warrant the expense involved by putting every case of measles on a prophylactic, even when no evidence of bacterial infection is present at the onset of the virus illness.

Historical

Haerem (1943) attempted to evaluate the effect of sulphonamides as antiviral rather than antibacterial agents in patients, probably mainly adults, admitted to a military hospital with measles. The dosage was 1 g. four times daily for four days. He found no significant differences in the duration of fever or the stay in hospital between treated and control cases, and he could find no previous mention of the use of sulphonamides in measles.

Begg *et al.* (1947) attempted to assess the total bacterial and haemolytic streptococcal counts in measles cases nursed in an L.C.C. hospital, and made a subsidiary study of the value of sulphadiazine in such cases. They concluded that sulphadiazine in a dose of 0.25 to 1 g. daily according to age was "of very doubtful prophylactic value," but were dissatisfied with their controls. The ages of the children are not given.

Badr El-Din (1951) attempted to assess the possible value of chlortetracycline ("aureomycin") "as an antiviral agent" in measles by attention to three things: (*a*) the increase or decrease of "catarrhal signs"; (*b*) the development and fading of the rash; and (*c*) the presence or absence of complications. The ages of only some, possibly the youngest, of the children are given. They were treated in hospital. Two out of 10 controls developed bronchopneumonia. No case of acute otitis media was reported, and no complications occurred in cases treated prophylactically, either with chlortetracycline or with combined penicillin and sulphadiazine. He found that chlortetracycline, given in the phase before the rash, diminished the catarrhal signs and also the intensity of the rash, if given early enough. He interpreted these as actions of chlortetracycline on measles virus, but other possible interpretations are discussed below.

No report has been traced of the use of sulphonamides or antibiotics in measles treated at home, nor of any attempt to assess the prophylactic value of these drugs in relation to the age of the child, to its past medical history, or to the presence or absence of bacterial infection at the onset of its measles. The present paper is an attempt to do this, as well as to define more clearly the limits of the problem, and to suggest what still remains to be done in any future, more extensive investigations.

Present Investigation

From December, 1950, to May, 1951, I attended 142 cases of measles in a rural partnership practice in the south-east of England, and the notes made at the time of seeing these patients, many of whom were treated prophylactically, mainly with sulphadiazine, have provided the material for this paper. As my notes were not then made with this particular research in mind, desirable details have sometimes not been recorded. Details of late complications after the children were out of the house from their attack of measles have not been analysed, as I was not then the only doctor seeing them. Particular care, however, was being taken during the epidemic to determine what signs of bacterial infection were present in each child at the onset of measles, and what evidence arose later in the illness to show that bacterial invasion had occurred. The details in this respect are therefore as accurate as circumstances allowed, and were all observed and recorded by me.

In defining the virus illness for myself, I took as its onset the first day of fever or typical symptoms, such as coughing or sneezing; and the end as the day when the rash had faded from the abdomen. At this stage a child was allowed up in the house, and outside on the next suitable occasion; but it was not returned to school or other crowds until three weeks after the onset of the rash, so as to minimize the risk of bacterial infection for that time. The episode which appeared to throw open the air passages of the head and chest to bacterial infection was the rash, first in the form of Koplik's spots. As the rash faded, so the mouth and eyes visibly healed, and one pictured the whole epithelium of the head and chest gradually recovering its resistance. Each of these stages in the virus illness might be defined more exactly in terms of changes in the leucocyte count, as is suggested below.

Information about the drugs used is incomplete in respect of dosage. The system I used for prophylaxis as a rule was a dose of tablets daily that totalled one more than the child's age in years, up to a total of six, in divided doses—that is, a child of 2 would have three tablets (each 0.5 g.) daily, in halves, while a child of 5 or over would have six tablets daily, one at a time. The duration of prophylactic treatment was from the day the child was first seen until at least the day the rash began to fade from the legs, sometimes until the rash had completely faded from the abdomen. I usually gave out the tablets at the time of my visit, working on this basis.

*Based on the Sir Charles Hastings Clinical Prize Essay, 1954.

Results

The first case was seen on December 3, 1950, and the last on May 25, 1951. Table I shows that the peak of the epidemic came in February and that the proportion of children on prophylactic drugs rose to a peak then and fell away again.

TABLE I

	Dec.	Jan.	Feb.	Mar.	Apr.	May	Total
On antibacterial prophylaxis	7	15*	38	25*	3	2	90
Not on drugs	13*	11	5	6*	2	4	41
On treatment for complications	1	1	2	2	1	0	7
On gamma-globulin	1	1	0	2	0	0	4*
Record of drugs missing	0	0	1	3	0	0	4
Total cases	21	27	46	36	6	6	142

* The four cases on gamma-globulin are shown in two places.

In later tables two cases on gamma-globulin alone are excluded from those receiving antibacterial prophylaxis, being shown as among those "not on drugs." Aspirin in some form was used for most children at the height of the fever. Details of the drugs used in the 90 cases receiving antibacterial prophylaxis are as follows: sulphadiazine alone, 74 cases; sulphadimidine alone, 10; "sulphatriad" suspension alone, 2; injection of penicillin alone, 1; sulphadiazine+sulphapyridine, 1; sulphadiazine+gamma-globulin, 1; sulphadimidine+sulphatriad suspension+gamma-globulin, 1.

Ages of Children Affected.—The epidemic began among the older schoolchildren of one village towards the end of term. From these it spread to their families and to other villages. Table II shows how, by February, it was the youngest schoolchildren and the older pre-school children

TABLE II.—*Monthly Distribution of Cases by Age Groups (The Number on Prophylactic Drugs is Shown in Parentheses)*

Age	Dec.	Jan.	Feb.	Mar.	Apr.	May	Total
1–	1	1 (1)	0	5 (3)	2 (1)	0	9 (5)
2–	3 (2)	2 (2)	1 (1)	8 (4)	1 (1)	0	15 (10)
3–	1	4 (4)	6 (4)	7 (4)	2 (1)	1	21 (13)
4–	1	3 (3)	4 (4)	3 (2)	0	1 (1)	12 (10)
5–	1 (1)	4	9 (8)	1 (1)	0	2	17 (10)
6–	2	3 (2)	10 (8)	1 (1)	0	1 (1)	17 (12)
7–	5 (3)	5 (2)	3 (2)	2 (2)	0	0	15 (9)
8–	3 (1)	1 (1)	6 (6)	3 (3)	0	0	13 (11)
9–	0	1	0	4 (3)	1	0	6 (3)
10–	2	1	2 (1)	2 (2)	0	0	7 (3)
11–	2	1	3 (3)	0	0	0	6 (3)
12–20	0	0	1	0	0	0	1 (0)
Over 20	0	1 (age 29)	1 (1) (age 26)	0	0	1 (age 21)	3 (1)
Total	21 (7)	27 (15)	46 (38)	36 (25)	6 (3)	6 (2)	142 (90)

who were being most heavily attacked. Not until March was the brunt of the attack borne by the youngest children at home. During April and May, which included the Easter holidays, the epidemic spent its force on the non-immune population.

Selection of Cases for Drug Prophylaxis

During December, and especially during January, as will be seen from Table II, drug prophylaxis was given particularly to the youngest children on the basis of the teaching in textbooks, as it was among them I expected the worst complications. Price (1947) states: "Measles is most fatal to infants and young children, 70% of the mortality occurring in children under 3 years of age." Gunn (1951) confirms this view, as follows: "Case fatality is highest under 1 year of age, but the greatest number of patients die in the second year because morbidity is much higher at this period." The two youngest children infected in this epidemic were each 14 months.

Children under 3.—The number of cases in infants was too small to allow more than a tentative opinion to be formed; but the impression gained from this outbreak was that if bacterial infection was absent, prevented, or adequately treated in children under 3 years of age, who had been breast-fed and whose mothers had had measles, the illness due to the virus infection was less severe than in older children of, for example, 10–12 years, some of whom were amongst the most prostrated, even in the absence of complications. The mildness of measles in a group of children between 8 months and 3 years, to whom Badr El-Din (1951) gave chlortetracycline in the stage before their rash, he attributed to an action of the drug on the virus itself; it may, however, have been due solely to the antibacterial effects of this drug. The duration of benefit derived from maternal immunity should be studied again among a larger number of the very young.

Other "Poor-Risk" Groups.—In addition to any which might occur in very young children, complications were also expected to be particularly prevalent among two other groups—namely, those who started their measles with evidence of bacterial infection already upon them, and those who, though evidently free of such infection, had a bad past history of naso-bronchial catarrh.

Evidence of Bacterial Infection Present at the Onset of Measles

The nature and severity of any signs of bacterial infection present in every child examined, whether for measles or not, was being recorded in detail at that time even though no symptoms referable to these signs were mentioned. From those records the Tables which follow have been prepared. The term "acute chest" has been used in the sense of Fry's (1953) paper, but limited to conditions that are usually febrile. "Subacute chest" includes the afebrile cases and others with minimal abnormal signs.

In Table III the incidence and severity of such preliminary bacterial infection is shown, followed by details of the actual conditions allotted to each degree of severity.

TABLE III.—*Monthly Incidence and Severity of Bacterial Infection Present at Onset of Measles*

	Dec.	Jan.	Feb.	Mar.	Apr.	May	Total
Total No. of cases	21	27	46	36	6	6	142
No. showing signs at onset	3	4	15	20	0	2	44
% showing signs at onset	14%	15%	33%	55%	—	30%	31%
Severe	1	1	4	5	0	0	11
Moderate	1	2	6	9	0	2	20
Mild or doubtful	1	1	5	6	0	0	13

In the 11 severe cases the following conditions were present: both drums very red, 4 cases; one drum very red and "subacute chest," 3; one drum very red, 2; bronchopneumonia, 1; "acute chest" and symptomless otitis media, 1.

In the 20 moderate cases were found: both drums injected, 4 cases; one drum reddened, 3; one drum opaque, healing, or old otitis media, 4; one drum with red malleolus, 2; both drums injected, one or both eyes sticky, 2; one drum reddened and "subacute chest," 1; one drum injected and mucopus in throat, 1; both drums injected and chronic tonsillitis, 1; tonsils exuding pus, 1; "subacute chest," 1.

The 13 mild or doubtful cases included: one drum slightly or partly injected, 7 cases; earache but drums not reddened, 2; both drums slightly injected but with good light reflex, 1; one meatus and drum reddened by heavy rash, 1; grossly enlarged tonsils but no exudate, 1; doubtful "moist sounds" in chest, 1.

In case the differences in severity of bacterial infection might merely be because the patients were seen early or late in the virus illness, the records have been analysed to show the stage reached by each case and the severity when the signs were first seen. This has been done in relation to "R" day—the day the rash appeared; "R minus" includes those seen before, and "R plus" those first seen after the rash appeared.

Table IV demonstrates that the severe infections were not necessarily seen late in the virus illness and confirms that these children were entering their measles already harbouring in varying degree the often symptomless seeds of possible bacterial complications.

TABLE IV.—*Severity of Bacterial Infection in Relation to Date of Rash*

	Time First Seen			Total
	R−	R	R+	
Severe	4	5	2	11
Moderate ..	7	7	6	20
Mild	3	7	3	13

Table III also reveals that the peak of preliminary bacterial infections was reached slightly later than the peak of the measles epidemic; but the relative incidence of severe, moderate, and mild bacterial infections did not materially alter with the change of season. The need, therefore, for an effective antibacterial prophylactic was greatest at the height of the bacterial rather than of the virus epidemic. This fact was not appreciated at the time, and Table V gives the monthly distribution of cases receiving antibacterial drugs in relation to the stage of their virus illness.

TABLE V.—*Cases Receiving Antibacterial Prophylaxis in Relation to "R" days, by Months*

	Dec.	Jan.	Feb.	Mar.	Apr.	May	Total
Total No. of cases ..	21	27	46	35	6	6	142
Prophylaxis:							
Starting on "R minus"	1	2	14	5	0	0	22
Starting on "R" ..	2	10	18	15	1	0	46
Starting on "R plus"	4	3	6	5	2	2	22
Total	7	15	38	25	3	2	90
% of monthly total ..	33	59	83	78	50	30	

It shows that the proportion of cases treated prophylactically more than doubled between December and February and that prophylactic doses of antibacterial drugs were being given earlier and to a slightly higher proportion of children at the peak of the measles epidemic in February than at the peak of the bacterial epidemic in March. The use of these drugs was evidently aimed more at preventing the complications of measles than at treating any bacterial infections which were found incidentally, though undoubtedly the latter factor was an influence.

"Catarrhal" Children

There were a number of children affected by measles in this epidemic who had suffered from recurrent previous attacks of respiratory infection often enough to merit being called "catarrhal" children. The number of such children seen is set out in Table VI, showing whether they presented with signs of bacterial infection as well as measles.

TABLE VI.—*Monthly Incidence of Bacterial Infection at Onset of Measles Among Normal and Catarrhal Children*

	Dec.	Jan.	Feb.	Mar.	Apr.	May	Total
Total cases	21	27	46	36	6	6	142
Normal children ..	17	22	32	22	6	5	104
With bacterial infection at onset { No.	2	2	8	7	1	0	20
With bacterial infection at onset { %	12	9	25	32	20	—	19
Catarrhal children { No.	4	5	14	14	0	1	38
Catarrhal children { %	19	19	30	39	—	20	27
With bacterial infection at onset { No.	1	2	7	13	—	1	24
With bacterial infection at onset { %	—*	—	50	93	—	—	63

Severity of These Infections

	Severe	Moderate	Mild	Total
Normal	4	6	10	20
Catarrhal	7	14	3	24

* No percentages were calculated for a total of fewer than six.

Table VI shows that in this epidemic the incidence of signs of bacterial infection at the onset of measles was three times as great in catarrhal children as in children with normal histories; and a higher proportion of those infections in the catarrhal children were severe or moderate, as opposed to mild.

As the catarrhal state should be considered in relation to age, Table VII gives the age distribution of these infected children.

The numbers involved in Table VII are too small for conclusions to be drawn, but there is a suggestion of two separate peaks in the incidence of the catarrhal state; one

TABLE VII.—*Ages of Catarrhal Children and Number Showing Signs of Bacterial Infection at Onset*

	1–	2–	3–	4–	5–	6–	7–	8–	9–	10–	11–	12–20	Over 20	Total
All children ..	9	15	21	12	17	17	15	13	6	7	6	1	3	142
Catarrhal:														
No. ..	1	2	4	5	3	4	6	6	3	2	2	—	—	38
%	10	13	19	42	18	24	40	46	50	30	30	—	—	27
No. infected	1	2	4	3	1	2	3	4	2	2	0	0	0	24
% infected*														63

* No percentages were calculated for a total of fewer than six.

comes in the 4–5 age group, at the end of home contact before school begins, and the other in the 7–9 age group after several years of school contact. If this were shown to be generally true, it might be that the catarrhal state is brought about, at least in part, by undue contact with carriers of bacterial infection, at first in the home and later at school. Mawson *et al.* (1953) noted a variation by age groups with a peak at 6 years, in the incidence of acute otitis media, among children treated at hospital, and suggested that, after going to school, "exposure of the child to an alteration in its usual bacterial environment plays a part in the causation of this disease." This is a subject that would repay further research.

Complications Resulting from this Epidemic

Table VIII sets out the number of children who were free of infection at onset, and the percentage of these who at a later visit showed signs of bacterial infection, complicating their measles.

TABLE VIII.—*Monthly Total of Children Free of Bacterial Infection at Onset of Measles, and Percentage of these Developing Complications Later*

	Dec.	Jan.	Feb.	Mar.	Apr.	May	Total
Total clear at onset ..	18	23	31	16	6	4	98
No. of these developing complications .	5	6	8	4	2	0	25
% with complications ..	28	26	26	25	30	0	26

One should, however, differentiate between children with normal and catarrhal past histories in estimating the risk of complications arising in any particular child who is free of signs of bacterial infection when starting measles. This is done in Table IX.

Part A shows the high percentage of children with normal past histories who were clear of signs of bacterial infection at the onset of measles; more than three-quarters of these remained clear. On the other hand, it was found that the proportion of catarrhal children who started their measles clear diminished steeply from February onwards, and of all those who started clear one-half later developed signs of bacterial infection during their virus illness.

Badr El-Din (1951) reported that chlortetracycline, given in the phase before the rash, diminished the catarrhal signs of measles. He attributed this to an effect of the drug on the measles virus, but it is more probable that the effect was an antibacterial one. This raises the question of what abnormal signs in the chest in measles can be attributed wholly to the action of the virus and which are the results

TABLE IX.—*Number of Complications Arising During this Epidemic of Measles*

A. Among those who were clear of signs of bacterial infection when first visited for measles, but who showed signs later

	Total Cases	Clear at Onset			
		No.	%	Showing Signs Later	
				No.	%
Normal histories ..	104	84	79	18	21
Catarrhal „ ..	38	14	37	7	50

B. Among those with signs of bacterial infection when first visited for measles, showing the number in whom these signs were changed

	Total Showing Signs at Onset	Signs After Measles			
		Aggravated	I.S.Q.	Reduced	Not Noted
Normal histories ..	20	3	5	11	1
Catarrhal „ ..	24	6	2	11	5

of any concomitant bacterial infection. Should any moist sounds, for example, be expected in the chest of a child with measles who is quite free from bacterial infection?

Part B of Table IX reveals at first sight a puzzling result—namely, that among 20 normal and 24 catarrhal children who started measles with signs of bacterial infection already present about half in each group showed a diminution or clearance of these signs during their virus illness. In discussing Table V, however, it was suggested that the presence of bacterial infection at onset had undoubtedly influenced the selection of cases for prophylactic treatment. This possibility is analysed in Table X, which shows separately, for both a pre-existing bacterial infection and a catarrhal history, how these factors influenced the selection of cases for prophylaxis.

It was shown in Table VI that signs of bacterial infection at onset were three times as common in catarrhal as in normal children during this epidemic. In Table X there is greatest

TABLE X.—*Distribution of Prophylactics Among those With and Without Preliminary Bacterial Infection or a Catarrhal Past History*

	Bacterial Infection at Onset		Past History	
	(1) Absent	(4) Present	(2) Normal	(3) Catarrhal
Total	98	44	104	38
No. on prophylactics ..	56 (57%)	34 (79%)	64 (62%)	26 (68%)

similarity between group 1 and group 2 with regard to the proportion on prophylactic drugs, while group 3 lies somewhere between those two and group 4, which had the largest proportion of children on prophylactics. In other words, the influence of selection for antibacterial prophylaxis was acting more strongly when I saw a case of measles in a catarrhal child with bacterial infection at the first visit, and was least active when I was dealing with normal children showing no early abnormalities.

The anomaly in Part B of Table IX now becomes understandable. Among those with signs at onset, whether with normal or catarrhal histories, such signs abated or disappeared in about half from each group because they were being given drugs in doses which were curative rather than prophylactic. The figures are too small for conclusions, but there is a further slight suggestion in part B of Table IX that, among catarrhal children, more often than among normal children, if the signs already present did not diminish, they were more likely to be aggravated than to remain *in statu quo* as a result of an attack of measles. In them the curative treatment had been insufficient.

What was the fate of those other children who were given drugs prophylactically, but who had shown no signs of bacterial infection when first examined for their measles? This touches the crucial problem of prophylactic versus curative treatment. The results obtained in this epidemic are set out in Table XI. This Table shows an unexpected result: proportionately more fresh late bacterial infections were seen among those on prophylactic drugs than among those not taking any. In point of fact, 19 out of these 25 fresh late infections occurred during December to February, before the peak of the "bacterial infection at onset," shown in Table III.

TABLE XI.—*Number of Complications in those who had no Signs of Bacterial Infection at the Onset of Measles; With Reference to Prophylactic Drugs*

Part I

	On Drugs	Not on Drugs	Record Incomplete	Total
Total clear at onset ..	56	41	1	98
No. with complications ..	17 (30%)	8 (20%)		25 (26%)

Part II.—Summary of Complications

	On Drugs	Not on Drugs
Acute otitis media	2	—
Symptomless otitis media	5	4
"Acute chest"	3	—
"Subacute chest"	3	—
"Acute" or "subacute chest" plus otitis media	1	1
Eyes abnormally affected	2	3
Nasal sores	1	—
Total	17	8

One possibility certainly is that by giving sulphadiazine or similar drugs to otherwise healthy children at the start of measles one was lowering their resistance to bacterial infection, should this reach them during the period of their rash. I have heard an eminent consultant paediatrician express the opinion that, since measles produces a leucopenia and since sulphonamides tend to depress the white-cell count, therefore sulphonamides should never be given to children during measles. While this may well be wise advice, it is possibly not the whole explanation of the anomalies revealed in Table XI, for one would then expect more evidence of infection in these treated children at the peak of the bacterial epidemic. Furthermore, it was shown in Table IX that in some children sulphonamides had exerted a beneficial action during measles.

Another possibility is that during the early months of the epidemic one was examining the children more closely. If this were true it could perhaps explain the larger proportion of cases noted during the first three months, but the overall difference in complications rate would still remain to be explained.

This difference between the complications rate of prophylactically treated and untreated children becomes even more striking if the children are divided into those with normal or catarrhal histories, in order also to test whether there had been any unconscious selection of bad-risk cases among those on prophylactics. This difference is shown in Table XII.

TABLE XII.—*Complications Rate in Prophylactically Treated and Untreated Children with Normal or Catarrhal Histories*

	Total	Clear at Onset				Complications			
		No.	No. Untreated	On Drugs		Untreated		On Drugs	
				No.	%	No.	%	No.	%
Normal histories	104	84	36	48	57	4	11	14	29
Catarrhal histories	38	14	5*	8	57	4	90	3	40

* One record of drugs incomplete.

Of those clear at onset, 57% were given prophylactic drugs, whether they were catarrhal or not; there was evidently no increased tendency for catarrhal children to be given these drugs, so long as they were free of signs of bacterial infection at the time measles began (see discussion of Table X). Table XII also reveals that, whereas sulphonamides given to those who showed no signs of bacterial infection when they first attended for measles may have prevented complications among catarrhal children, these same drugs appeared to increase considerably the risk of complications occurring in normal children.

The distribution of complications among the age groups was studied to see whether there was any difference in this respect between treated and untreated children.

Table XIII shows that in the untreated group the three severe or moderate complications all occurred in younger children than did the five mild complications; while in the treated group there were relatively more severe or moderate complications, and the 11 which occurred were at both younger and older ages than the six mild ones.

TABLE XIII.—*Number and Severity of Complications by Ages Among Prophylactically Treated and Untreated Children*

	1–	2–	3–	4–	5–	6–	7–	8–	9–	10–	11–	12–20	Over 20	Total
Untreated:														
Complication:														
Sev.+mod.	—	—	1	—	1	1	—	—	—	—	—	—	—	3
Mild ..	—	—	—	—	—	—	3	1	1	—	—	—	—	5
Nil ..	5	2	6	—	5	4	2	1	1	1	3	1	2	33
On drugs:														
Complication:														
Sev.+mod.	3	—	—	2	—	1	2	1	1	—	1	—	—	11
Mild ..	—	—	3	—	1	1	1	—	—	—	—	—	—	6
Nil ..	—	6	3	5	6	7	2	6	—	1	1	—	1	38
Record incomplete ..	—	1	—	—	—	—	—	—	—	—	—	—	—	1

Discussion

In this epidemic the action of sulphadiazine appears to have been twofold. When given in near-curative doses to children, especially catarrhal children, who showed signs of active bacterial infection at the start of their measles, the drug acted beneficially by reducing the severity of the bacterial infection in about half the treated cases, while the remainder had probably received an insufficient dosage; but when sulphadiazine or another sulphonamide was given to normal children, apparently free of bacterial infection at the start of their measles, the drug appeared to confer no benefit, but rather to increase the rate and severity of complications as compared with similar untreated children.

If these findings were to be confirmed by other observers and in a larger series of cases, the whole question of the use of sulphonamides in any disease associated with leucopenia—for example, also influenza—would have to be re-examined. It might be possible to do serial white-cell counts throughout a sufficient number of cases of measles and influenza to determine the incidence and degree of leucopenia in relation to bacterial complications, both in prophylactically treated and in untreated cases. Penicillin and sulphonamides could be compared as antibacterial prophylactics in diseases associated either with leucopenia or with leucocytosis. It may be that when bacterial infection is already present at the onset of such a virus illness there is already a sufficient leucocytosis to offset any later drop in the white-cell count, due either to the superimposed virus infection or to drugs. In such cases sulphonamides may not be so harmful, though they are not necessarily to be preferred to penicillin.

Puckey (1941) reported a hospital case in which granulopenia had developed after a leucocytosis due to pneumonia, treated with sulphapyridine; measles and infective hepatitis developed during the treatment. At the end of the pneumonia, after ten days on sulphapyridine and three days after the measles rash appeared, the white-cell count was 25,000 per c.mm. In spite of stopping sulphapyridine the white-cell count dropped steadily to 4,600 per c.mm. (24% neutrophils) on the fourteenth day after the rash. How much of this leucopenia was due to sulphapyridine and how much to measles and infective hepatitis it is not possible to judge, but the question is one which undoubtedly needs to be examined again.

Conclusions

A fresh investigation of the problem of antibacterial prophylaxis in measles should be carried out soon by those with access to a large number of cases treated at home, bearing in mind the following conclusions which can be drawn from the present report of an outbreak in a country practice of 142 cases of measles, 90 of which were given prophylactics.

(1) The complications rate in untreated normal children during this outbreak was 4 out of 36 (11%), whereas in untreated catarrhal children it was 4 out of 5 (80%).

(2) Among all children, irrespective of age or past medical history, who were free of signs of bacterial infection at the onset of their measles, the complications rate was higher in those given sulphonamides prophylactically (17 out of 56=30%) than in the untreated (8 out of 41=20%).

(3) Some children already showed signs of bacterial infection when their measles began. This occurred in 24 out of 38 (63%) of catarrhal children, but in only 20 out of 104 (19%) of normal children.

(4) Twice as many such infections, already present, were graded as moderate or severe as opposed to mild in the catarrhal children, as compared with those in normal children.

(5) There was a seasonal variation in the amount of preliminary bacterial infection, especially among catarrhal children. In this outbreak the peak of such bacterial infection fell in March, though the peak of the measles epidemic passed during February.

(6) There was no evidence that the severity of the virus illness of measles was unduly marked in children under 3 so long as bacterial infection was absent; in fact, the impression was gained that such children suffered less from their measles than did some of their older siblings.

(7) There was evidence that the sulphonamides given had been acting curatively rather than prophylactically in those children who started measles showing signs of preliminary bacterial infection; but that in about half of these the dose had been insufficient.

(8) There was some evidence to suggest that the catarrhal state of a child was not something permanent, but that it might vary according to undefined circumstances both before and after the age of starting school.

(9) Since it appears from an analysis of the present observations that sulphonamides are of value to some children, but not to others, in preventing bacterial complications of measles, a further investigation is needed into the incidence, severity, and duration of any changes in the white-cell count of normal and of complicated measles, in both treated and untreated children, some receiving sulphonamides and some penicillin. Such an investigation might reveal ways of defining stages of the virus illness of measles in terms of leucopenia and the consequent risk of bacterial complications.

(10) Such an investigation, if also carried out in other diseases associated with leucopenia and apt to be followed by bacterial complications, such as influenza,

might further reveal a general principle of value in determining the choice of antibacterial drugs for prophylactic or curative use, especially in virus infections.

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Note.—Since this essay was presented, the College of General Practitioners has begun a two-year investigation into the value of sulphonamides and antibiotics in the prophylaxis of the bacterial complications of measles. The conclusions reached in this essay were made available to the College, which hopes to obtain records of 2,000 to 3,000 cases of measles by the end of 1955.

THE EARLY DIAGNOSIS OF SPINAL TUMOURS

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The first patient to be successfully treated for spinal tumour was a British Army officer who was operated on by Sir Victor Horsley in 1887 (Gowers and Horsley, 1888). He recovered from paraplegia and lived for twenty years before he died from an unrelated condition. Since this demonstration that at least some spinal tumours were surgically accessible, and indeed curable, it has been of the utmost importance to diagnose such neoplasms early in their course and to locate them accurately.

It is not the purpose of this paper to describe in detail the established clinical syndromes, excellent accounts of which already exist (Elsberg, 1941). Rather is it intended to make use of the opportunity afforded the Service medical officer of having patients under observation from an early stage, and to comment on the clinical picture and diagnosis at this stage. Failure to make an early diagnosis may lead to incomplete recovery or even permanent disablement, despite the removal of a benign tumour, due to prolonged cord compression.

This paper is concerned with primary and secondary tumours which involve the spinal cord or nerve roots; such lesions develop in the vertebrae or in the structures within the spinal canal. The material for the investigation consists of 36 verified cases of spinal tumour treated between January, 1940, and December, 1951, at the Military Hospital for Head Injuries, Oxford. During this period there were approximately 1,150 admissions with spinal symptoms, 813 bearing the label of prolapsed intervertebral disk. These cases were drawn from the three Services and therefore represent a special cross-section of the population in regard to age and sex. There were 32 males and 4 females, the ages ranging from 17 to 60 years, 30 of them being under 35 years (see Table III).

Pathology

The lesions met with in this series are given in Table I. The frequency of the histological groups may be compared with those in a civilian series of 275 cases reported by Elsberg (1941). Table I reveals two important differences between the two series in the relative incidence of these tumours. There are no meningiomas and there is a high proportion of metastases in the military series. These points require further comment.

TABLE I.—*Pathology*

Present Series		*Civilian Series* (Elsberg, 1941)	
Tumour	No. of Cases	Tumour	No. of Cases
Neurofibroma	15 (41·6%)	Meningioma ..	73 (26·5%)
Metastatic	8 (22·2%)	Neurofibroma ..	64 (23·2%)
Vascular	4 (11%)	Sarcoma	31 (11·2%)
Ependymoma ..	3 (8·3%)	Glioma	14 (5%)
Glioma*	3 (8·3%)	Metastatic ..	14 (5%)
Osteoclastoma ..	2	Ependymoma ..	11 (4%)
Melanoma	1	Vascular	9 (3·3%)
		Others	59 (21·5%)

* Two histologically unverified.

Meningiomas are found most often in middle-age, and when they occur in the spine women are affected twice as often as men (Learmonth, 1927); this group of patients not being represented in the section of the population from which our cases are drawn thus accounts for their absence in this series.

Metastatic tumours occurred in no less than 22% (8 out of 36) of the patients in this series, mainly young adults; the oldest patient with a secondary tumour was 42, four patients were in the thirties, one was 19, and two were 17 years of age. However, the incidence of metastatic spinal tumours is doubtless underestimated in civilian series based on statistics from surgical explorations: many overtly metastatic tumours, frequently presenting in the terminal stages of malignant disease, never reach the neurosurgeon. Nevertheless, it is important to appreciate that a metastatic neoplasm cannot be ruled out on age alone, nor on the apparent absence of a primary growth.

In six patients (Cases 29–34) the histological sections of the spinal tumour revealed secondary anaplastic carcinoma, suggestive of bronchial origin. Although the presence of a primary bronchial carcinoma was later confirmed by a post-mortem examination in only one instance (Case 34), the available facts favoured a bronchial origin for the primary growth in all six cases. Pulmonary or hilar shadows were present in four of the five patients in whom *x*-ray films were available.

Another patient with a secondary neoplasm (Case 35) died elsewhere, and a post-mortem examination revealed anaplastic secondary carcinomatous deposits in the spine, liver, lung, and iliac glands; a primary tumour was not found. The final patient in this group (Case 36) had vertebral collapse associated with a tumour in the lower end of the humerus, the radiological diagnosis being "osteogenic sarcoma"; no further information about the pathology is available.

The proved or probable bronchial origin of six of the eight metastases indicates the importance of *x*-raying the chest of every patient suspected of having a spinal cord tumour. A carcinoma of the lung may, however, remain clinically latent or not be apparent radiologically for some time in the presence of spinal metastases (see Case 31, described below). Occasionally the primary growth may be very small and be revealed only after a careful search at the post-mortem examination (Turner and Willis, 1938).

Although we have emphasized the frequency of metastatic growths it should be stressed that essentially operable and benign tumours were present in 40% of the Service cases and in 48% of the civilian series. It is in these cases that early diagnosis is so important.

Symptoms

With primary spinal tumours the symptoms usually have a gradual onset, preceding physical signs by many months. As some 70% of them are situated posteriorly or posterolaterally in the spinal canal it is natural that sensory should precede motor disturbances as a rule. Sphincter disorders usually occur later.

With secondary neoplasms, however, the course is characteristically rapid, paraplegia often developing within days of the first symptoms. In two cases in this series this interval was less than 10 days, whilst the longest history given by a case of secondary tumour was three months. All eight patients with metastatic lesions were unable to walk on admission, while this advanced picture was seen in only 6 of the 29 patients with primary spinal neoplasms. The rapid advance of some of these metastatic growths is illustrated by the following case.

Case 31. Extradural Metastatic Tumour with Three Weeks' History.—A trooper in the Life Guards, aged 17, was perfectly well until three weeks before admission, when he began to have backache. Pain down the right leg developed twelve days later, and three days before admission he rapidly developed a complete flaccid paraplegia below L 4 Plain *x*-ray films of the spine showed only some doubtful erosion of the pedicle of D 11, but lumbar puncture revealed a manometric block and a cerebrospinal fluid protein of 1,000 mg. per 100 ml.; myelography showed the block to be at D 11. Laminectomy exposed an extradural tumour which was shown histologically to be a metastatic carcinoma, suggestive of bronchial origin. A chest *x*-ray film on the day of operation was clear. Later, supraclavicular, axillary, and intercostal glands became involved and a subcutaneous tumour which appeared over the scapula showed on biopsy the same appearances as had the spinal mass. The patient was transferred later to another hospital, where he was said to have developed cough and dyspnoea with signs of pulmonary collapse three weeks before his death, which followed three months after the laminectomy. A post-mortem examination was not made.

In contrast, the following case report illustrates the gradual onset of symptoms due to a benign spinal tumour.

Case 9. Dorsal Neurofibroma with Two Years' History.—An A.T.S. woman aged 26 first noticed numbness and a burning sensation in her right leg some two years before admission. For seven months she had experienced pain in the lower back and in both calves. During the four months before admission her gait had become increasingly unsteady, and for two months there had also been urgency and frequency of micturition. Examination revealed increased tone and weakness of the left leg, a sensory level at D 8, and bilateral extensor plantar responses. A spinal *x*-ray film showed erosion of the pedicle and scalloping of the body of D 7. Lumbar puncture revealed an almost complete spinal block and a cerebrospinal fluid protein of 120 mg. per 100 ml. She made a good recovery following removal of neurofibroma attached to the left seventh dorsal nerve root.

Pain is the commonest symptom, but it is not a constant feature. It occurred in 29 cases of this series (81%), which agrees with the findings of Craig (1936), who recorded pain as being present in 80% of 300 cases from the Mayo Clinic. Not uncommonly, and especially with tumours of the cauda equina, there may be partial or complete temporary remissions of this symptom. Such remissions were found in five of our nine cauda equina tumours, and Allen (1930) reported remissions in 25% of 69 patients with tumours in this region.

Two types of pain are experienced : root pain and dull central backache. Root pain, which is the more common, was a manifestation in 24 of our cases (66%), and in 19 patients (53%) it was the initial symptom. That root pain is not confined to extramedullary tumours was pointed out as long ago as 1914 by Pearce Bailey. Indeed, such pain was found in nearly half the intramedullary growths studied by Kernohan and his colleagues (1931). In two of the five intramedullary tumours in the present series there was root pain, but in each case motor symptoms had preceded this by many months.

Root pain has certain characteristics. It may be aching or lancinating, is referred to the appropriate dermatome, and may be so severe as to require morphine for its relief. It is often accentuated, and sometimes precipitated, by raising the intrathecal pressure, as by straining and coughing; this is probably due to an alteration in the relationship of the tumour to near-by roots. It may be worse at night and be relieved by walking about or sleeping in a chair. Eaton (1941) suggests that this may be due to the lengthening of the spine which he has demonstrated experimentally to occur in recumbency. It is thought that the disks increase in thickness on lying down and that this is responsible for the pain by altering the relationship between the roots and surrounding structures.

Dull backache is common as a symptom of cauda equina tumours, but so frequently is this a complaint of other disorders that few sufferers should be suspected of having a spinal tumour on this account alone. It is backache which persists and fails to respond to rest that should arouse suspicion. Moreover, in the presence of a spinal tumour it is commonly accompanied by root pain; in only 4 of the 22 patients with backache in the present series was this the only type of pain. Though most constant in cauda equina tumours, backache is often a distressing feature of metastatic deposits in the vertebrae. It may even occur in intramedullary tumours, as evidenced by two patients in this series.

Pain was not a feature in seven patients (19%). This agrees with the figure of 20% reported by Craig (1936) and of 25% by Spurling and Mayfield (1936). The type of lesion present in our cases without pain varied, but included three examples of vascular malformation. There were two neurofibromas, one extradural secondary carcinoma of the bronchus, and one intramedullary tumour.

In other cases sensory disturbances apart from pain may be predominant, such as a sensation of tingling, pins-and-needles, burning, or numbness. These are common as early manifestations which occasionally occur alone, but more frequently are associated with backache and root pain.

Loss of power usually follows sensory disturbances, but it may occur as an initial symptom, and this is especially likely to happen with intramedullary tumours. In three of our cases this led to a diagnosis of poliomyelitis.

Sphincter Disturbance.—Although complete retention or incontinence of urine or faeces is a late manifestation, increasing constipation or some difficulty in micturition is not an uncommon symptom in patients with spinal tumour. Apart from 10 paraplegic patients who had incontinence or retention on admission, nine patients had experienced some difficulty in micturition, but in none was this a presenting feature.

Physical Signs

The presence of suspicious symptoms, such as root pain and persistent backache, warrants a thorough examination of the whole patient. In the majority of cases with spinal tumour this will yield further evidence of such a lesion. The *spine* is tested for local tenderness or deformity, muscle spasm, and limitation of movement. Spinal tumours in the lumbar region—that is, of the cauda equina—commonly

produce erector spinae spasm, a stiff spine, and even scoliosis. The range of straight-leg raising and the effects of jugular compression and of coughing are recorded. Careful examination of the *nervous system* will often indicate the level of the lesion fairly accurately, especially if the patient is paraplegic. Weakness, wasting, reflex changes, and sensory loss all contribute to this localization. The *general examination* should include in particular a search for the cutaneous pigmentation or tumours of neurofibromatosis and for angiomata of the skin or retina; and palpation of the abdomen, rectum, and breast for evidence of primary malignancy. The chest is *x*-rayed for primary or secondary neoplasms and to visualize any thoracic extension of dumb-bell tumours of the spine.

Dandy (1925) claimed that over 90% of tumours could be both diagnosed and located in this way, but he was referring to the established picture. Such signs may be minimal if the patient is seen at an early stage, and indeed in some cases when special investigations give definite evidence of a tumour. These investigations normally consist of plain *x*-ray films of the spine, lumbar puncture, and myelography. There were five cases in this series with minimal signs, and they are mentioned later in relation to the differential diagnosis from prolapsed intervertebral disk, which they all closely resembled. One may be noted here as an example.

Case 4. Neurofibroma of the Cauda Equina with Minimal Neurological Signs.—A major aged 60 complained of pain in the left buttock and thigh, accentuated by coughing and sneezing, for eleven months. He had massage and short-wave diathermy, following a diagnosis of fibrositis and osteoarthritis. Six weeks before admission he developed low lumbar pain. There was no weakness or sensory or sphincter disturbance even to direct questioning. The only signs on admission were a stiff lumbar spine, a slight depression of the left knee-jerk, and slightly impaired sensation, to pin-prick only, on the outer side of the left thigh. A preliminary diagnosis of prolapsed intervertebral disk was made. Plain *x*-ray films were normal, but a lumbar puncture showed a cerebrospinal fluid protein of 360 mg. per 100 ml., and myelography suggested a possible tumour at L 4. At operation a neurofibroma was removed from the left side of the cauda equina. He made a good recovery.

Though not a feature of any of our cases there may be no physical signs whatsoever, even when investigations reveal a tumour (Cohen and Kaplan, 1943; Toumey *et al.*, 1950). In all these reported cases, together with the five patients in this series with minimal signs, the tumour was situated in the cauda equina. Here the bony canal is relatively roomy, and this may be the explanation for the paucity of signs.

Ancillary Investigations

Radiography

Plain X-ray Films.—In 16 cases (44%) plain *x*-ray films of the spine showed appearances suggestive of spinal tumour. There may be gross vertebral destruction (three cases), but, more frequently, less well-marked changes occur such as erosion of the pedicles and laminae (10 cases), widening of the intervertebral foramina (three cases), and scalloping of the posterior aspect of the vertebral bodies (three cases). The value of such films is obvious, and in addition the changes produced by a primarily vertebral tumour or other spinal disease may be revealed.

Myelography, using ethyl iodophenylundecylate ("myodil"), was employed in the final localization of the tumour in 22 cases. Although myelography is by no means always necessary for the detection of a spinal tumour, it is invaluable in accurate localization, and may be the only way in which a neoplasm can be excluded in suspicious cases. Owing to oedema and secondary vascular effects the clinical level may be false, and at the best the clinician has only clues concerning the extent of the lesion. The myelogram may also suggest whether the neoplasm is intramedullary or extramedullary, the longitudinal extent of the lesion, or the presence of multiple tumours. It also gives the surgeon the opportunity to mark the lesion in relation to the spinous processes pre-operatively and so render exposure more precise.

Lumbar Puncture

This procedure revealed an abnormality in every case in the present series. The changes were partial or complete manometric block, xanthochromia, and raised cerebrospinal fluid protein, though all three features were not always present.

Manometry.—Partial or complete manometric block was found in 26 of our cases (76%). So important is the detection of spinal block that manometry must be carefully carried out in every case. In patients with suspected tumour it has been our practice, where possible, to have two observers—one to apply bilateral jugular pressure and the other to watch the manometer. First, light compression of the neck is applied, as this may reveal an incomplete block, past which fluid could be driven by firm compression (Stookey *et al.*, 1925); normally a rise of 10–30 mm. is to be expected at this stage. Then firm pressure is applied for ten seconds, and this should, in the absence of a block, produce a steady rise up to 300 mm. with a more rapid fall on release. If there is a complete block there will be no rise at all, whilst any definite delay in either the rise or the fall indicates an incomplete obstruction. The application of a sphygmomanometer cuff to the neck with the pressure raised to approximately 80 mm. may be used as an alternative to manual jugular compression.

The patient must be warned to relax during the manometry, and in particular reassured about the neck compression, since if, as is natural, he reacts to it by holding his breath and tensing his abdominal muscles, this alone will produce a rise in the manometer reading even in the presence of a complete block. If no obstruction was demonstrable at the beginning of the test then manometry should be repeated after the removal of a few millilitres of fluid for analysis, before the needle is withdrawn.

It has been shown experimentally by Poppen and Hurxthal (1934) that manometric abnormalities are not to be expected until the diameter of the remaining patency of the spinal subarachnoid space is less than the diameter of the lumbar puncture needle used. Occasionally a block will be evident only after the removal of fluid, possibly due to the meninges collapsing down on to the tumour (Ayer, 1920). Repeated dry taps suggest obliteration of the subarachnoid space by a large cauda equina neoplasm; the needle may have entered the neoplasm.

Cerebrospinal Fluid Protein.—The cerebrospinal fluid was examined in 35 cases, and all showed a rise of protein from 50–6,000 mg. per 100 ml. (upper limit of normal taken to be 45 mg. per 100 ml.). Of the four cases with slight increases only (50–65 mg.), three had manometric evidence of block, so that the significance of the slight abnormality was not likely to be overlooked. Whilst the increase in protein is usually the result of a spinal block, it may occur in the absence of such obstruction, and is then thought to be due to exudation from the surface of the tumour. Raised protein from this cause may thus be found in those cases of cauda equina tumour in which the cerebrospinal fluid is withdrawn *above* the site of the neoplasm. Of the nine cauda equina tumours reported here, five were associated with over 1,000 mg. of protein per 100 ml.

Xanthochromia.—This was present in 16 of the 29 cases in which the appearance of the fluid was recorded. Whilst usually associated with a very high protein, whether below a block or above a cauda equina tumour, there was one case with xanthochromia but only 110 mg. of protein per 100 ml., and two others with 150 mg.

Cells.—In most patients there was no increase of cells in the cerebrospinal fluid, but in seven cases a slight increase in lymphocytes was reported (7–12 per c.mm.). This feature gave no hint of the type of lesion present, since it was found in cases of neurofibroma, angioma, glioma, and secondary carcinoma.

In view of the obvious diagnostic value of lumbar puncture, the question arises of how readily, or at what stage, should one recommend it. Two factors influence the

decision. The first is the appreciation that a spinal block may exist, due to a tumour, when neither plain *x*-ray films nor clinical examination of the central nervous system have shown any abnormality—or nothing diagnostic (see Case 20, described below). The second is the possible risk of the procedure, fear of which may dissuade investigators outside special units from pursuing their cases to this vital stage. During the past two years in this hospital over 2,000 diagnostic lumbar punctures have been performed safely without sequelae other than headache in a few cases. However, there are some cases reported from time to time of the precipitation or aggravation of the neurological signs after puncture in cases of spinal cord tumour. Because of this possibility, Horrax (1947) has suggested that lumbar puncture should be carried out in cases of suspected cord tumour only when there are facilities for proceeding, if necessary, to immediate laminectomy.

From a survey of our own experience and of cases in the literature, we think that the risk of lumbar puncture has been exaggerated, with the important exception of cervical cord tumours in which puncture may precipitate respiratory paralysis. In such suspected cases, and also in rapidly progressive cases, it is advisable to postpone this examination until the patient is in a special unit. Apart from these instances, we hold the view that if a patient complains persistently of backache or root pain for which there is no obvious cause, and the films of the spine are normal, a lumbar puncture should be performed, as this may be the only way by which a spinal tumour may be excluded with confidence. So common are these symptoms, and so uncommon are spinal tumours, that there clearly will be numerous investigations of this kind which will come to nothing, and it would be quite impracticable to restrict lumbar punctures to neurosurgical units.

The value of lumbar puncture is exemplified in Cases 3 and 20.

Case 3. Neurofibroma of the Cauda Equina; Minimal Neurological Signs.—A flying officer aged 22 gave a history of an attack of left-sided sciatica aggravated by coughing for a period of a fortnight, nine months before admission. The pain recurred during a bumpy car journey three months prior to admission, and two days later he went to bed for an unrelated condition (duodenal ulcer). While in bed the left-sided sciatica became worse, and a month before admission he also developed sciatic pain on the right. On examination he walked stiffly and there was slight weakness in both legs. No sensory loss was demonstrated and the reflexes were symmetrically brisk. Straight-leg raising was reduced to 20 degrees on each side. Plain films were normal, but lumbar puncture revealed partial block with xanthochromic fluid containing 950 mg of protein per 100 ml. A myelogram showed a block at L 1. A cystic neurofibroma was removed from the cauda equina and he made a good recovery.

Differential Diagnosis

It is clear that in most cases the diagnosis of an intraspinal lesion is easy once the suspicion is aroused, although its exact nature may require special study. However, it will be apparent from the symptomatology that in the earlier stages, and in other cases in which neurological abnormalities remain minimal, a spinal tumour may readily be confused with other conditions. Some of the difficulties are well exemplified in the Services, where in general the patients come under medical observation early.

The average delay between first consulting a doctor and undergoing operation was 6.4 months in the present series. On the other hand, reports from civilian centres point to much longer delays; in one series the corresponding interval was seventeen months (Van Wagenen and Rossier, 1938). Specialist medical advice is particularly easy of access in the Services, and this probably accounts for the shorter delay. Late operations are due largely to the multiplicity of common conditions (see Table II) which can give rise to the same types of pain, and to the considerable rarity of spinal tumours. Added to this is the not infrequent paucity of abnormal signs in the central nervous system even in many long-standing cases. In the majority of cases there is for some time no specific neurological disability to draw attention to the central nervous system, whilst even in retrospect it is difficult to discern any single characteristic of the pain complained of, other than its persistence, that should have led to the suspicion of a spinal tumour as its cause. In short, we wish to stress that there is often a period of many months in the natural history of a spinal tumour when confusion with other conditions may readily arise, with consequent delay in surgical treatment.

TABLE II.—*Some Incorrect Diagnoses in 28 Patients*

Diagnosis	Cases
Fibrositis, rheumatism	10
Prolapsed disk, sciatica, neuritis	10
Functional, hysteria, neurosis	9
Disseminated sclerosis	5
Tuberculous spine	4
Poliomyelitis	3
Peripheral neuritis	2
Syringomyelia	2
Subacute combined degeneration	1
Neurosyphilis	1
Cervical rib	1
Renal colic	1
Peptic ulcer	1
Total	50

Late diagnosis may prove disastrous to the patient with a benign spinal tumour. Irreparable damage may occur either from prolonged compression of the cord or from interference with its blood supply, so that even if a total removal is still possible recovery is slow and perhaps incomplete.

Although the possible differential diagnoses are legion certain conditions are more commonly simulated than others.

Prolapsed Intervertebral Disk

This diagnosis was made at some stage in 10 cases of the present series. The five patients already mentioned with minimal signs in the central nervous system closely simulated disk lesions, and two of them have been described (Cases 3 and 4). A further example illustrates the difficulty in diagnosis (Case 20).

Case 20. Ependymoma of the Cauda Equina Resembling Prolapsed Intervertebral Disk.—A man aged 28 had attacks of back pain for twelve months before operation. These would last five or six days and resolve with physiotherapy. A month before operation right-sided sciatica complicated an attack and he came into hospital. His pain increased in bed, but the only abnormal physical signs were flattening and rigidity of the lumbar spine and slight scoliosis, straight-leg raising was limited to 60 degrees bilaterally and there was a doubtful depression of the knee- and ankle-jerks on the right. In this hospital the diagnosis after *x*-ray examination, but before lumbar puncture, was of prolapsed intervertebral disk, as had been thought elsewhere. Further investigation showed, however, a complete spinal block and xanthochromic fluid containing 2,500 mg. of protein per 100 ml. A myelogram revealed a block at L 1, and at operation an ependymoma of the cauda equina was partially removed.

Two not infrequent features of tumour cases may render the simulation of the disk syndrome very close: (1) Temporary remissions of pain, which may even coincide with treatment applied for a supposed disk lesion and so be taken as confirming the suspicion. (2) Signs of disordered spinal mechanics—stiff spine with diminished straight-leg raising.

Although in most cases the history and evolution of the clinical picture are quite different in the two conditions, in some cases it may be impossible to make the distinction clinically, and Case 4 shows the value of lumbar puncture in settling the problem. Lumbar puncture is not everywhere a routine procedure before disk surgery is undertaken, but it would seem to be a wise precaution in the light of this experience. It has always been the practice in this centre.

"Backache"

There were 10 cases diagnosed at some stage as "fibrositis" or "rheumatism"—that is, non-articular rheumatism—on account of the complaint of backache alone. One of the cases (Case 20) had attacks of backache as the only symptom for eleven months, and, furthermore, these bouts

TABLE III.—*Service Patients with Spinal Tumours*

Case No.	Sex and Age	Duration of Symptoms	Main Symptoms	State on Admission	Lumbar Puncture			Plain *X*-ray Spine	Pathology
					Colour	Block	Protein mg./100 ml.		
1	M 32	8 years	Back and root pain (sciatic)	Minimal signs	Y	B	6,000	+	Neurofibroma, cauda equina
2	M 27	11 months	Back and root pain (to testicle)	Slight weakness left leg	Y	B	1,600	−	" " "
3	M 22	9 "	Back and root pain (sciatic)	Minimal signs	Y	P.B.	950	−	" " "
4	M 60	11 "	" " "	" "	N	Reproduced pain	360	−	" " "
5	M 20	9 "	" " "	" "	Y	P.B.	3,000	−	" "
6	M 22	5 "	Neck pain and weakness of all limbs	Tetraplegia	?	P.B.	50, later 70	+	Neurofibroma, C 1–2
7	M 26	7 "	Weakness right leg	Mild paraparesis	Y	B	4,200	−	Neurofibroma, T 12
8	F 22	2½ "	Back and root pain (to chest). Weakness legs	Paraplegia	N	P.B.	960	−	Neurofibroma, T 7
9	F 26	22 "	Back and root pain (to both calves)	Slight weakness left leg	?	P.B.	120	+	" "
10	M 19	3½ "	Neck and root pain (to shoulder). Weakness all limbs	Tetraparesis	N	B	55	+	Neurofibroma, C 5
11	F 31	6 "	Root pain (sciatic)	Weakness right leg	Y	B	1,200	+	Neurofibroma, cauda equina
12	M 28	16 "	Numbness and weakness legs	Mild paraparesis	N	B	65	+	Neurofibroma, C 7–T 1
13	M 36	13 "	Root pain (to abdomen). Weakness of legs	Flaccid paraplegia	Y	B	1,000	+	Neurofibroma, T 9
14	M 19	3 "	Weakness legs	Paraparesis	?	B	130	−	Neurofibroma, T 6–7
15	M 23	6 "	Progressive weakness all limbs	Tetraplegia	Y	N	300	−	Neurofibroma, cervical
16	M 45	3 years	Weakness leg	Weakness leg	N	P.B.	200	−	Angioma, cervical
17	M 24	22 months	Root pain (to abdomen)	" "	Y	N	150	−	Angioma D 12
18	M 60	18 "	Numbness and weakness legs	Mild paraparesis	N	N	80	−	Angioma D 9–11
19	M 33	5 "	" " "	Flaccid paraplegia	N	N	220	−	Angioma D 10–11
20	M 28	12 "	Back and root pain (sciatic)	Minimal signs	Y	B	2,500	−	Ependymoma cauda equina
21	M 29	7 "	Root pain (sciatic)	Weakness leg	Y	N	150	−	" " "
22	M 20	7 "	Back and root pain (sciatic)	Paraparesis	Y	B	Clotted	+	" " "
23	M 23	2 "	Back and root pain (sciatic). Weakness legs	Paraplegia		Not done		+	Osteoclastoma L 4
24	M 19	8 "	Back and root pain (to chest)	Mild paraparesis	N	N	80	+	Osteoclastoma D 8
25	M 36	5 "	Weakness legs	Paraplegic	N	B	80	−	" Intramedullary tumour " D 10–12
26	M 31	8 "	Back pain. Weakness leg	Weakness leg	N	N	55	+	"Intramedullary tumour" D3
27	M 22	3 "	Back pain. Weak legs	Paraplegic	Y	B	2,000	−	Spongioblastoma D 2–3
28	F 22	7 "	Neck pain	Mild tetraparesis	Y	N	110	−	" Primary malignant melanoma " C 3 root
29	M 35	3 weeks	Back pain. Weak legs	Paraplegic	N	B	300	−	Anaplastic secondary car. D 4. ? Bronchogenic
30	M 17	9 days	Root pain (to chest). Unable to walk	"	N	B	200	+	Anaplastic secondary car. D 1–3. ? Bronchogenic
31	M 17	3 weeks	Back and root pain (to leg)	"	?	B	1,000	+	Anaplastic secondary car. D. 12 ? Bronchogenic
32	M 19	5 "	Root pain (to chest and arms). Unable to walk	"	Y	P.B.	240	+	Anaplastic secondary car. C 7. ? Bronchogenic
33	M 42	3 months	Back and root pain (to chest). Weakness legs	"	Y	B	350	−	Anaplastic secondary car. D. 10. Bronchogenic
34	M 30	10 days	Unable to walk	"	?	P.B.	120	−	Secondary car. D 7–10. Bronchogenic (P.M.)
35	M 31	3 weeks	Back and root pain (to thighs). Unable to walk	"	N	B	150	+	Secondary car. D 7. Unknown primary (P.M.)
36	M 35	7 "	Back and root pain (to legs). Weak legs	Paraparesis	?	B	600	+	" Secondary osteogenic sarcoma " D 8

Y = Yellow. N = Normal. B = Block. P.B. = Partial block. + = Abnormal. − = N.A.D. P.M. = Post Mortem

responded to physiotherapy. Case 1 illustrates that a long history cannot exclude tumour even when pain in the back remains the principal complaint.

Backache, especially if there is restricted spinal movement with muscle spasm, may raise the possibility of disease of the spine itself such as tuberculosis, ankylosing spondylitis, and osteoarthritis. A plain *x*-ray film usually disposes of these diagnoses.

Case 1. Neurofibroma of Cauda Equina—Prolonged Complaint of Backache without Neurological Disability.—A man of 32 was admitted in 1943 with a history of intermittent backache since 1935, when he had fallen, injuring his back. On that occasion he recovered after three weeks in bed, but two months later backache recurred and pain radiated down the left leg into the calf. There was no relief of symptoms in spite of five months in hospital, and he returned to work. In 1940 he joined the Army and, although able to manage a twenty-five-mile route march, he could not do physical training, and in 1942 "collapsed" with pain in his back during an assault course. Examination in hospital on several occasions did not reveal any obvious organic causes. On admission here he complained of backache and pain down the left leg which was continuous and aggravated by coughing. There were no other symptoms. Examination showed minimal sensory loss over the lateral aspect of the left thigh, slight diminution of the left knee-jerk, and an abnormal plantar response; no weakness could be detected. Straight-leg raising was normal, but jugular compression aggravated the pain. There was spasm of the erector spinae muscles with some limitation of spinal flexion. *X*-ray examination of the spine revealed some flattening of the pedicle of L 3. Manometry demonstrated a complete spinal block, the cerebrospinal fluid being yellow and containing 6,000 mg. of protein per 100 ml. Myelography showed a complete obstruction at the level of L 3. At operation, eight years after the onset of symptoms, a neurofibroma was removed from the third left lumbar nerve root. He made a good recovery.

In this story there was nothing about the pain, apart from its persistence, which was suggestive of a tumour. The neurological signs, even after eight years, were not gross.

Visceral Diseases in the Thorax or Abdomen

Root pain is produced by many types of visceral disease, and this pain may be indistinguishable from that produced by irritation of the same roots by spinal neoplasm. Not uncommonly, abdominal operations of varying severity have been carried out for persistent pain before the correct diagnosis of spinal tumour has been made (Spurling and Mayfield, 1936; Craig, 1936).

Case 2. Neurofibroma of the Cauda Equina Resembling Visceral Disease.—An Army driver aged 27 had increasing pain in the left loin, radiating to the testicle and the front of the thigh, for eleven months; the pain was accentuated by coughing. It was thought to be of renal origin, but when intravenous pyelogram and urinary examinations were repeatedly negative he was referred to a psychiatrist, who diagnosed prolapsed intervertebral disk. The patient was transferred to this hospital, where examination showed slight weakness and depression of reflexes in the left leg and some diminution of sensation below L 1 segment.

Lumbar puncture revealed a complete spinal block and a yellow fluid containing 1,600 mg. of protein per 100 ml. Myelography confirmed the block, and removal of a neurofibroma from the second lumbar nerve root completely relieved the symptoms.

Non-neoplastic Diseases of the Spinal Cord

In a few cases of neurological disease such as disseminated sclerosis, poliomyelitis, transverse myelitis, and motor neurone disease, clinical examination may suggest a focal spinal lesion, and if the cerebrospinal fluid protein is elevated the presence of a tumour may be suspected. It is in such cases that resort to myelography may be necessary. There were no cases in the present series in which a diagnosis of disseminated sclerosis was made after admission to this hospital. However, this is a common initial diagnosis in spinal cord lesions, and in these cases a lumbar puncture should always be carried out.

The importance of examining the blood and cerebrospinal fluid for the Wassermann reaction may be mentioned here; in relation to this it is interesting to remember that the original description of the Froin syndrome probably referred to three cases of syphilitic meningomyelitis (Greenfield and Carmichael, 1925).

Extradural Abscess

This is commonly simulated only by metastatic tumours, but the resemblance can be close. In this series three of the eight metastatic tumours were initially thought to be extradural abscesses, and emergency laminectomy was performed in one case (No. 34).

Case 34. Extradural Metastasis Resembling Extradural Abscess.—A man of 30, who had been treated for secondary syphilis three months previously, developed increasing weakness of the legs over three days. Examination showed a spastic paraparesis with pyramidal reflex changes, and a diagnosis of syphilitic meningomyelitis was made. Spinal *x*-ray films were normal, but a lumbar puncture showed a partial block with 120 mg. of protein per 100 ml. and 5 cells per c.mm. Paraplegia was rapidly progressing, and a neurosurgeon was consulted. A diagnosis of extradural abscess was made and an emergency laminectomy performed. A mass of pathological tissue was found extradurally which was said to be suggestive of a healing abscess, both macroscopically and microscopically. Two months later chest *x*-ray examination showed a hilar mass; only "congestive changes" at one base had been seen on a film a week before operation. Death occurred three months after laminectomy, and post-mortem examination showed a primary bronchial tumour with widespread secondary deposits, including those in the spinal dura.

These cases, then, illustrate the difficulties besetting the clinician confronted with a patient suffering from a spinal tumour. Two points emerging from a study of this series are, firstly, the value of lumbar puncture, and, secondly, the necessity for exploring the possibility of an intraspinal lesion in puzzling cases of backache or root pain and of periodic examination of the nervous system in search of minimal signs.

Treatment

Laminectomy should be carried out as soon as the diagnosis is established and the location determined. Complete removal of the tumour is, of course, the ideal procedure and is usually possible in cases of neurofibroma. Of the 14 neurofibromas operated on in this series all were totally resected and 11 made a complete recovery. The only unoperated case in the whole group was that of a dumb-bell fibroma with a large extraspinal mass in the neck, and the patient succumbed within nine weeks of admission (Case 15). The remaining tumours were only partly removed or else were left with decompressions, and the majority were then treated with radiotherapy. Incomplete removal followed by radiotherapy gave a satisfactory immediate recovery in two cases of ependymoma of the cauda equina. There was one operative death due to aspiration pneumonia. No long-term follow-up has been possible.

Conclusions

Spinal tumours in their early stages often present a difficult diagnostic problem. This is partly because the presence of such a lesion may not be even thought of owing to its comparative rarity, and partly because it may simulate other more common disorders for many months or even years before there is total cord compression.

A variety of conditions may be confused with spinal tumour, the most frequent being "rheumatism," prolapsed intervertebral disk, neurosis, disseminated sclerosis, and visceral disease within the thorax or abdomen. However, a careful evaluation of the type of pain and a full neurological examination will in most cases of spinal tumour suggest the possibility of such a lesion. Furthermore, diagnosis is often possible, at a stage when neurological signs are minimal or debatable, by performing a lumbar puncture. Less often, plain *x*-ray films of the spine lead to detection.

Surgical treatment is often completely curative, but only if the diagnosis is made before irreversible pressure changes have occurred in the cord or nerve roots.

Summary

Service patients tend to come under observation early, and this opportunity has been taken to comment on the early clinical picture and diagnosis of spinal tumours based upon a study of 36 cases at the Military Hospital for Head Injuries, Oxford.

The average delay between the first consultation and operation was 6.4 months, which is shorter than in most civilian series. Common diagnostic errors were "fibrositis" or "rheumatism" in 28% of cases, prolapsed intervertebral disk or "sciatica" in 28%, and "neurosis" in 25%.

The most frequent complaint was backache and/or root pain, occurring in 81% of cases; in 61% it was the initial symptom. In five cases there were only minimal or doubtful neurological signs, although pain had been present for nine months or more.

The most frequent neoplasm was the neurofibroma (41%). An unexpectedly large number of metastatic tumours was present (22%), most of which appeared to be of bronchial origin; three of these tumours occurred in patients between 17 and 19 years of age. A metastatic lesion should be suspected if the history is short and the clinical picture advanced.

Reference is made to the ancillary investigations, and attention is drawn to the value of lumbar puncture in early diagnosis, especially in doubtful cases. Evidence of spinal block was obtained in 76% of cases, and in all 35 patients whose cerebrospinal fluid was examined the protein content was raised.

Lumbar puncture may be the only way of excluding a tumour with any confidence in patients with persistent back or root pain for which there is no obvious cause. The possible dangers of the procedure are negligible, except in rapidly progressive cases and those with suspected cervical tumours, the investigation of which should be delayed until neurosurgical aid is available.

We are indebted to the medical officers responsible for the detailed clinical notes, often made under considerable wartime difficulties, without which this study could not have been possible. We have received much encouragement from members of the staff of the Nuffield Department of Surgery, Oxford, and are particularly grateful to Mr. Joe Pennybacker for suggestions and to Dr. Peter Daniel for help with some of the pathological data. Above all, our gratitude is due to Mr. Walpole Lewin for much helpful advice and criticism at all stages in the preparation of this paper. We wish to thank the War Office for permission to publish these cases.

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COARCTATION OF THE AORTA AND PREGNANCY

REPORT OF FIVE CASES

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Coarctation of the aorta is an uncommon finding in adults and shows its higher incidence in males in a proportion of 3 males to 1 female (Abbott, 1928). The association of this anomaly with pregnancy must therefore be of some rarity. Benham (1949) was able to find records of only 53 cases in the literature and added three personal reports. To these can be added the following cases: Dieckmann (1941), 4; Magnin (1944–5), 1; Novak (1947), 1; Clark and Koenig (1947), 1; Cohen (1949), 1; Marcorelles (1949), 1; Money (1949), 1; Sundfør (1950), 3; Vander Veer and Kuo (1950), 2; Brown (1950), 4; Greig and Parker (1951), 1; Bond (1951), 1; Kerr and Sodeman (1951), 5; Soulié *et al.* (1951), 3; Notter (1952), 1; Miller and Falor (1952), 2; Stéphan (1952), 1; Peterson *et al.* (1953), 1; and Taylor and Pollock (1953), 1; making a total of 91 recorded cases.

In view of the infrequency of pregnancy complicating coarctation of the aorta the assessment of the risks involved and the prognosis and management of any individual patient become considerations of great importance and difficulty for the physician and obstetrician whose experience of this association of conditions must necessarily be limited. It would seem desirable that as many records as possible be reported so that a firmer basis for judgment may be provided. With this in view the following five cases are reported.

Case 1

The patient, aged 25, was referred from the local antenatal clinic at the third month of her first pregnancy for the diagnosis of a suspected heart lesion. A "rheumatic heart" was said to have been discovered when she was 5 years old, and from about the age of 10 she had had symptoms very suggestive of mild intermittent claudication. These lasted on and off for about five years. The only symptom of note after this had been mild dyspnoea on exertion, which she thought was probably a little more prominent since the beginning of her pregnancy. Except for this and very occasional "bilious attacks" (vomiting, mild headaches, but no eye symptoms) she was perfectly fit and well.

Examination showed the classical signs of coarctation of the aorta. Blood-pressure readings were: right arm, 170/100; left arm, 150/95; right leg, 120/100; left leg, 115/90. The heart size was within normal limits. She had a mild ichthyosis, mostly involving the skin of the trunk, upper arms, and thighs, and this had been present since childhood. She had no other abnormal physical signs. *X*-ray examination of the chest showed a poorly developed aortic knuckle and notching of the third, fourth, sixth, seventh, eighth, and ninth ribs of both sides. Blood counts, blood urea, and Wassermann reaction were normal. Intravenous pyelography studies and an electrocardiogram were within normal limits. The amylobarbitone test resulted in an average blood pressure of 130/95 compared with 155/100 before the test.

She attended the medical clinic regularly and the pregnancy proceeded uneventfully. At the 27th week a slight generalized enlargement of the thyroid gland was noted. She was admitted to hospital at the 36th week for a final rest prior to confinement. One week later she began to complain of nausea, hot flushes, palpitations, and precordial pain. Over the course of three hours her pulse rate rose from 80 to 115 a minute, the systolic blood pressure from 135 to 165 mm. Hg, and the diastolic pressure from 95 to 100 mm. Hg. It was decided to perform caesarean section at once. The lower segment operation was done under spinal analgesia, and a living infant weighing 5 lb. 6 oz. (2.4 kg.) delivered. The patient was not in labour and there was much bleeding from the thickened lower segment of the uterus. It was noted that the abdominal aorta was half normal size. The puerperium was uneventful, and she was discharged from hospital after three weeks.

The patient was seen again at the medical clinic three months later. She had remained fit and well with no complaints except for slight dyspnoea on exertion, which was no worse than before the pregnancy. There were no notable changes in the physical signs. She remained in good health for five months after delivery. One night, about one and a half hours after going to bed, she awoke, told her husband she felt "queer," rapidly became unconscious, and died within half an hour. A post-mortem examination was not carried out.

Case 2

A primipara aged 21 was referred from the local antenatal clinic for diagnosis of a suspected heart lesion. She had had no serious illnesses as a child and had played games and cycled as other girls of her age. She had no complaints of headaches or pains in the legs. Eighteen months previously she had been off work for three months with dyspnoea on exertion, lethargy, and ankle oedema. These symptoms returned when she became pregnant, but on stopping work she regained her former good health.

On examination she was found to be 20 weeks pregnant. The blood pressure in the arms was 165/85 mm. Hg, and the systolic pressure in the legs, by palpation of the dorsalis pedis artery, 105 mm. Hg. A systolic murmur could be heard at the base of the heart at the level of the second rib on the left side close to the sternum. A loud systolic murmur was heard over the whole posterior chest, maximal in the mid-zone on both sides. A systolic thrill was felt in the left supraclavicular region. *X*-ray examination showed the heart to be normal in size and configuration, though the aortic knuckle was not seen. Rib notching was not present.

The pregnancy continued uneventfully until the thirty-eighth week, when she was admitted with uterine haemorrhage. A central placenta praevia was diagnosed and a living infant of 5 lb. 14 oz. (2.7 kg.) delivered by lower segment caesarean section. No note was made of the size and pulsation of the abdominal aorta. Except for a mild pyrexia and anaemia the puerperium was normal. On the tenth day the cardiac bruits were unchanged. No collateral circulation could be felt or seen. The blood pressure in the arms was 150/75 mm. Hg, while that in the legs could not be determined. The femoral artery pulsations were not felt.

Case 3

A woman aged 43 was referred to the local maternity hospital because of a raised blood pressure, upper abdominal pain, and malaise three weeks before her fifth child was due. She had had four previous normal pregnancies and labours, and her past medical history was uneventful.

On examination, except for a blood pressure of 130/90 mm. Hg, no abnormalities were noted. The urine was free from albumin. For the next two weeks she did not complain, had blood pressures of 150/90 and 140/98, and the urine remained albumin free. She was admitted to hospital at term complaining of headache and vomiting, her blood pressure was 130/90, and a very faint trace of albumin was found in the urine. She was treated conservatively and the blood pressure varied from 140/90 to 170/110 with no albuminuria. One week after admission, with the blood pressure 190/130, labour was induced surgically. The next day her blood pressure was 120/70 and she was noted to be "bright and cheerful." In the afternoon she was allowed to walk around the ward in order to encourage the start of labour. Fifteen minutes later she dropped down dead.

Post-mortem examination showed the pericardial sac to be full of blood. Coarctation of the aorta was present at the origin of the left subclavian artery, and at this point the aorta measured ⅜ in. (1 cm.) across. The ascending aorta was dilated to a diameter of 3 in. (7.6 cm.), and showed a split in the intima with a dissecting aneurysm which had ruptured through the adventitia into the pericardial sac. The left ventricular muscle was slightly hypertrophied. A bicuspid aortic valve was present. The uterus contained a normal full-term foetus, and except for congestion of the lungs all other organs were normal.

Case 4

A 1-para 2-gravida aged 24 was walking through the backyard of her home carrying her 15-months-old baby on her arm when she suddenly collapsed, quickly lapsed into unconsciousness, and died after about 30 minutes. No further history was available.

Post-mortem examination showed a small ruptured aneurysm at the bifurcation of the right middle cerebral artery. The subarachnoid haemorrhage was greatest around the optic chiasma, the brain stem, and between the cerebrum and cerebellum. It had tracked along the sulci and was present in the ventricles too. The skull was intact and there were no injuries of the scalp. The heart was slightly enlarged owing to hypertrophy of the left ventricle. The mitral valve showed some fibrosis and scarring, while the aortic valve had two functioning cusps only. Stenosis of the aorta was present at the junction of the arch and descending portions, and measured ½ in. (1.3 cm.) at this point. The uterus contained a normally developed foetus 9½ in. (24.2 cm.) long, corresponding to the pregnancy of 20 to 22 weeks' gestation. Except for venous congestion the remainder of the organs were normal.

Case 5

A woman aged 30 had been attending her local antenatal clinic for her first pregnancy and was said to have enjoyed perfect health both before and during her pregnancy. At about the 38th week she suddenly collapsed at home and died within a few seconds.

On post-mortem examination the pericardial sac was distended with blood which had poured through a small rupture in the posterior wall of the root of the aorta. The ascending and transverse parts of the aorta were very thin and were dilated to a point just behind the left subclavian artery. Here the aorta became narrow and "completely obliterated," while below this point the vessel was noted to be "abnormally narrow." There was marked hypertrophy of the left auricular and left ventricular muscles. The aortic orifice was very narrow and would not admit one finger. The uterus contained a normal 38-weeks foetus, while all other organs were macroscopically normal.

Discussion

The comparatively small number of cases on record do not allow of any valid conclusions of a statistical nature to be drawn from them. Many of the reports are deficient in important details; some are only mentioned in textbooks and articles. Again there is a tendency to report only the unusual and dramatic, and therefore no detailed statistical analysis is possible if an imbalanced picture is to be avoided.

Coarctation of the aorta in an adult carries with it well-defined risks, and the association of pregnancy increases these hazards, especially of rupture of the aorta. There are records of 11 patients with the anomaly whose death was directly associated with pregnancy, and in 6 of them death was due to rupture of the aorta just before term or during labour. In 9 out of the 11 cases the coarctation remained undetected during the pregnancy. Earlier diagnosis, and therefore appropriate supervision and management, may have a large part to play in improving the prognosis. However, perusal of the literature shows that many women have successfully negotiated repeated pregnancies without any demonstrable adverse effects in spite of the presence of coarctation, which in many cases remained undetected until after the pregnancy. One woman of 56 (Weber and Price, 1912) had produced 11 children and died 12 years after her last pregnancy, and another of the same age (Strassmann, 1922) produced seven children. While not diminishing the seriousness with which this association of conditions has to be regarded, these facts suggest that a uniformly gloomy prognosis for all cases is unwarranted. The records, however, give no indication of those women with coarctation of the aorta who are likely to be unaffected by pregnancy—information which might be a useful guide in the management of future patients. Comparative studies of blood-pressure readings or the degree of collateral circulation appear to have no value in deciding prognosis in an individual patient.

Antenatal supervision of pregnancies is now an established practice, and it is usual to examine the chest at least on the first attendance and to measure the blood pressure frequently throughout the pregnancy. These are unusually favourable conditions for detecting those patients who have an existing coarctation of the aorta, and routine palpation of the femoral pulses in all patients with hypertension, however mild, would undoubtedly disclose further examples of the associated conditions.

The advice regarding management of these patients has varied according to the personal experience of the physician and his reading of the literature at the time. Some have advised that all cases should have therapeutic abortion with sterilization (Mendelson, 1940), some have advised caesarean section at term with or without sterilization (Walker, 1943; Baber and Daley, 1947; Brown, 1950), while others have allowed labour to proceed normally (Soulié *et al.*, 1951). Surgical treatment of coarctation of the aorta has not only stimulated early diagnosis of the condition but will now influence the management of pregnancy cases. The patients of Marcorelles (1949) and Peterson *et al.* (1953) were both operated on successfully during their pregnancies, and Miller and Falor (1952) describe the case of a woman who was operated on three months after her first pregnancy and successfully negotiated a second a year later.

It is suggested that all patients in whom these conditions coexist should be referred to a thoracic unit, where the possibility of surgical intervention might be considered. A pregnancy of less than 20 weeks' duration should, in itself, be no contraindication to operation. Until more experience is available it would be wiser to defer surgery if the diagnosis of the coarctation is first made in the later months of pregnancy. Management otherwise is, in general, similar to that applied to pregnant women suffering from rheumatic heart disease. Undue enlargement of the heart, signs of aneurysmal dilatation of the ascending aorta, or symptoms suggestive of cerebral aneurysm indicate the advisability

of terminating the pregnancy. A rise of blood pressure in the last few weeks of pregnancy or during labour should be viewed seriously and caesarean section undertaken.

Summary and Conclusions

There are records of only 91 patients in whom coarctation of the aorta was associated with pregnancy. Five further cases are reported.

The association carries with it serious risks, especially of rupture of the aorta, but, in spite of these, many women have not been adversely affected by multiple pregnancies and have lived to an age well beyond the average expectation of life in coarctation of the aorta. Earlier diagnosis and surgical treatment of the coarctation offer means of improving the prognosis.

Diagnosis is not difficult, and depends mainly on the awareness of the examiner and routine palpation of the femoral pulses in all patients with hypertension. The antenatal clinic offers unusual opportunities for early diagnosis.

General principles in management of these patients are described and the importance of individual assessment and surgical treatment for the coarctation is stressed.

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It is recorded in the annual report of the Chalfont Colony for 1953–4 that an arrangement has been made with the National Hospital for Nervous Diseases whereby patients, either before or immediately after entering the Colony, will go to the Queen Square or Maida Vale Hospitals for full investigation and report. The executive committee believes that a certain percentage of cases now going to the Colony will be intercepted, treated at Queen Square or Maida Vale, and returned to normal life in a few weeks. The medical staff of the Colony will also receive guidance from the staff of the National Hospital about the treatment of those patients who are finally admitted to the Colony.

HAEMORRHAGIC GANGRENE OF THE SKIN IN CHRONIC LYMPHATIC LEUKAEMIA

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In chronic lymphatic leukaemia both specific and non-specific skin lesions may occur, but distinction between the two often rests on the histological picture.

In a study of the dermatological manifestations of the lymphoblastoma–leukaemia group, Epstein and MacEachern (1937) found that out of 60 patients with chronic lymphatic leukaemia 27 (45%) had non-specific skin lesions. Of these, the haemorrhagic type was commonest (15 patients), and usually occurred in the terminal stages. Although Sutton and Sutton (1941) mention that purpura sometimes results in necrosis and ulceration of the skin and mucous membranes, we are unable to find any reference to the occurrence of haemorrhagic gangrene of the skin in chronic lymphatic leukaemia.

Case History

A 58-year-old male librarian was admitted on October 5, 1952, having been found unconscious in his garden. There was a past history of chronic bronchitis. A provisional diagnosis of subarachnoid haemorrhage was confirmed by lumbar puncture. During routine examination he was found to have moderate splenomegaly but no palpable glandular enlargement, and there were no other abnormal signs. A blood count showed: Hb, 92%; red cells, 4,680,000 per c.mm.; white cells, 12,900 per c.mm. (polymorphs 22%, eosinophils 0.5%, monocytes 1%, lymphocytes 76.5%); no primitive forms; platelets, 168,750 per c.mm.; E.S.R., 1 mm. in the first hour (Wintrobe); clotting-time, 6 minutes 30 seconds; bleeding-time, 3 minutes. A sternal marrow puncture showed a typical picture of chronic lymphatic leukaemia.

As it was uncertain whether the subarachnoid haemorrhage was in any way connected with the leukaemia, and as the leukaemia was symptomless, with the possible exception of being related to the subarachnoid haemorrhage, it was decided not to treat the patient at this stage. He was discharged in November, having made an uneventful recovery from the subarachnoid haemorrhage, and he was seen regularly as an out-patient.

In January, 1953, he was readmitted to hospital with a three-days history of fatigue with fever and the coughing up of sputum flecked with bright blood. On examination he was wasted and there were symmetrical localized areas of ecchymoses over both upper arms and both malar eminences. The only other abnormal physical finding was splenic enlargement, which was of the same degree as on the previous admission. A blood count at this time showed no marked change from the previous count, and the bleeding- and clotting-times were still within normal limits. The possibility of a leukaemoid reaction to generalized tuberculous infection was considered, and accordingly the sternal marrow was re-examined in February, 1953. No evidence of tuberculous histology was seen, and the appearance was still that of a lymphatic leukaemia. A chest *x*-ray examination showed extensive cavitation in the right upper lobe, but repeated sputum tests and examination of laryngeal swabs did not reveal any evidence of tuberculosis.

The skin lesions became more numerous. Each would start as a small patch of purpura which enlarged rapidly

and then developed superficial gangrene. Progressively and symmetrically they involved the pinnae, arms, forearms, buttocks, legs, and toes (see Fig.).

"Acthar gel," in a dose of 50 mg. 12-hourly intramuscularly, was begun, but two days later the patient went into coma and died on February 8.

Necropsy Findings.—External Examination: There was a dark-purple discoloration of ears and face, except for the circumoral area, with ulceration of both ears and cheeks. Dark-purple patches were present in the region of both nipples, on the outer side of both arms, with ulceration of the upper arms, on both buttocks, and on the outer side of both thighs and legs, with purpuric spots at the border. *Circulatory System:* A large pericardial effusion was present consisting of clear yellow fluid. *Respiratory System:* The lungs were emphysematous, with a cavity in the right upper lobe approximately 2 cm. in diameter, and an adjacent dark solid area with a hyperaemic border showing central softening. There was no macroscopic evidence of tumour. *Digestive System:* The liver was enlarged (weight 2,326 g.) and extensively infiltrated with pale semi-translucent nodules. These swelled above the cut surface, and were approximately 1 cm. in diameter. *Haemopoietic System:* Lymph nodes around the coeliac axis and at the hilum of the spleen and para-aortic nodes were prominent. Cut surface was mottled, with reddish areas. The spleen (weight 1,162 g.) showed a pale cut surface with prominent Malpighian bodies. *Nervous System:* An intact berry aneurysm arose from the anterior communicating artery of the circle of Willis. Adjacent to the aneurysm there was brownish discoloration of the arachnoid.

Photograph showing skin lesions in the early stages.

Histology.—Right Lung: An old infarct was present with central necrosis and peripheral haemorrhage and infiltration with lymphocytes and polymorphs. Elsewhere there were foci of lymphocytes. *Liver:* Lymphocytic infiltration had occurred and there were many deposits of irregular polygonal-celled trabecular carcinoma with a fine stroma. Mitoses were numerous. The appearances were those of metastases of an aplastic bronchial carcinoma. *Spleen:* Pulp spaces and sinusoids were distended with lymphocytes. *Coeliac Lymph Node:* Germ centres were no longer distinguishable, and the whole node was packed with lymphocytes of adult type. *Skin:* Both superficial and deep corium contained small recent haemorrhages, and in the same situation the veins contained organizing thrombi. The arteries appeared normal. There was no leukaemic infiltration.

Comment

Progress of the skin condition to extensive symmetrical gangrene had occurred as a terminal event, and it is difficult to assess the degree to which this contributed to the patient's death, particularly so when the double pathological condition found at necropsy is considered. Carcinoma, especially of the bronchus, is common nowadays, and chronic lymphatic leukaemia is by no means rare, so that the association of the two conditions in the case here reported is probably fortuitous. Nevertheless, only 27 cases of carcinoma with leukaemia had been reported up to 1950 (Berk and Movitt, 1945; Even, Vibert, and Oury, 1950). In our case no primary growth was found in the lungs or in any other organ, but the histological appearance of the deposits in the liver was suggestive of primary carcinoma of the bronchus.

We wish to thank Dr. A. R. Kelsall for permission to publish this case.

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THE EPIDEMIOLOGICAL APPROACH TO OBSTETRIC MEDICINE*

BY

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The annual report of a well-known teaching maternity hospital says that it had 1,413 admissions during the year 1950. These contributed 1,270 deliveries and 85 abortions. The stillbirth rate was 9.34 per 1,000 (an exceptionally low figure) and the neonatal death rate 17.32. In 31 tables are listed some of the features of numerous abnormal cases. How cases were chosen for admission and how they relate to the community as a whole are not indicated. The clinical detail presented is reduced to a bare minimum and there is no text or discussion whatever. The lay-out and scope of such annual reports are time-honoured, but make it very difficult to extract figures of scientific value. The time has surely arrived when all statistical reports should be drawn up according to the laws of epidemiology. Now that maternity services are organized on a regional basis, the leading hospital in an area might collate data from all sources and prepare reviews illustrating and discussing trends of morbidity and mortality in a defined population.

Maternity records are particularly suitable for systematic surveys because birth and death certification provides an automatic and accurate statistical framework for every administrative area. There is little difficulty, of the kind which arises in the study of chronic disease, in arriving at a biologically meaningful epidemiological "unit." A "maternity"—the cycle of pregnancy, childbirth, and recovery—is easily defined, and takes place in a limited and relatively constant period of time.

The group of maternities to be studied having been defined, the next step is to classify them. To reduce the population to a series of fairly homogeneous groups it is desirable to classify by legitimacy; by the maternity service to which a case has been booked, irrespective of the place of delivery; and perhaps by home address, if it is desired to limit the study to the residents of an area. Multiple maternities should be separated from single.

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Most analyses will be carried out on groups of single legitimate maternities, which should be classified by age and parity.

Influence of Maternal Physique and Health

Since reproduction is a physiological function it is reasonable to suppose that maternal physique and health will be an important influence. It is, unfortunately, very difficult to assess health objectively, but a social classification is a useful indirect measure of the way of life of patients which is highly correlated with health and physique. Even better and more simple, it is useful to classify mothers by height. Height is associated statistically with social class and with general health. As one moves down the social scale, the proportion of small women and of unhealthy women increases (Baird, 1952b; Baird and Illsley, 1953). It has been shown that mothers in different height categories experience different rates of foetal mortality, prematurity, and difficult labour (Baird, 1949, 1952a, 1952b). The theory is that mothers who have been brought up in a satisfactory environment will grow to the maximum stature permitted by their genetic constitution, and will be in good general health, whereas those brought up in poor surroundings will be stunted and less healthy.

The Epidemiological Method in an Aberdeen Inquiry

I propose to illustrate the use of the epidemiological method in obstetrics by describing an inquiry made in Aberdeen into the nature and means of preventing unexplained deaths among mature babies. Accounts in greater detail have been published by Baird, Walker, and Thomson (1954), Walker (1954), and Walker and Turnbull (1953).

Prematurity has for long been recognized as a major cause of "unexplained" neonatal mortality. It is not so widely known that many babies which are mature by birth weight—that is, over 5½ lb. (2.5 kg.)—die for no very obvious reason. In a classification of obstetric deaths (stillbirths plus first-week deaths) by clinical cause (Baird, Walker, and Thomson, 1954) we distinguished two "unknown" categories, classified in terms of birth weight into "premature, cause unknown," and "mature, cause unknown." In both, pregnancy and labour were clinically uneventful, and at necropsy only uninformative conditions such as "prematurity," "atelectasis," "anoxia," and so on were found, the underlying cause of death being unexplained. Of 1,008 consecutive obstetric deaths in Aberdeen Maternity Hospital booked cases during the 15-year period 1938–52, 13.7% were classified as "mature, cause unknown."

A comprehensive analysis of these deaths by age, parity, and other factors was undertaken. Since booked hospital cases, especially multiparae, are more or less selected (Baird and Thomson, 1954), a study based on all city maternities, whether delivered in hospital or not, would have been preferable. Information on a city-wide basis has become available only recently, and in a relatively small city obstetric deaths are not numerous enough to permit dependable statistical analysis unless the results of many years are pooled. All stillbirths (not first-week deaths) in the city since 1938 could be classified by clinical cause, but complete information on age and parity was not available. In the booked hospital series, the stillbirth and first-week death rates behaved in relation to age, parity, and social class in much the same way as comparable national statistics, and long experience has shown that booked hospital primiparae at any rate may be regarded as a representative sample of first maternities in general; it was therefore believed that the behaviour of the obstetric death rate was fairly typical of the child-bearing population as a whole.

The "mature, cause unknown" obstetric death rate was 6.3 per 1,000 total births in primiparae, 4.5 in second and third pregnancies, and 4.4 in fourth and later pregnancies. In primiparae, especially, there was a steady rise as maternal age increased—from 2.3 at age 15–19, to 19.0 at age 35 and over. Obviously elderly primiparae (say 30 and over) form a high-risk group for deaths in this category.

Since the data were compiled from a 15-year series, during which time the overall obstetric death rates had fallen considerably, trends in the "mature, cause unknown" obstetric death rate were investigated in relation to time. In the hospital series this rate behaved rather erratically, and certainly showed no sign of a consistent fall such as had occurred in the rate from all causes. Examination of data for the whole of Aberdeen showed that the "mature, cause unknown" stillbirth rate fell from just over 7 per 1,000 in 1939–40 to slightly under 5 in 1944–5. During 1946–8, the period of the post-war rise in the birth rate, when there were relatively more first births and an increase of average maternal age, the rate rose to the pre-war level, falling again in 1949–51 to about 5 per 1,000 (Baird, Thomson, and Duncan, 1953).

Examination of the hospital data for primiparae by social class and maternal height showed no consistent trends.

"Premature, cause unknown" death rates, on the other hand, show consistent trends in the expected direction by social class and height; the rates fell steadily during the war and showed no post-war rise; the gradient by age in primiparae is not so steep and steady as in the "mature, cause unknown" group, and very young as well as elderly mothers seem to have a relatively high death rate. Excess deaths in the "premature, cause unknown" group seem to be statistically associated with poor social class, small stature, and poor general health and nutrition of mothers; this is by no means obvious in the case of "mature, cause unknown" deaths. Though the two groups no doubt overlap aetiologically, the distinction by birth weight—above or below 5½ lb. (2.5 kg.)—seems to be fairly effective in separating deaths of different character.

Obstetric Death Rate in Post-maturity

Clinically, we had the impression that many "mature, cause unknown" deaths took place when pregnancy was prolonged past term (40 weeks) and that death was frequently preceded by foetal distress—that is, marked irregularity or slowing of the foetal heart, or the passage of meconium. To check these impressions statistically it was necessary to have very detailed and uniform notes on the duration of pregnancy and on the occurrence of foetal distress during labour. That recent hospital records sufficed for a fairly accurate analysis shows that an interest in epidemiology has had the useful effect of raising the standard of clinical recording in the hospital to a very high level.

It has been known for some time that the stillbirth and neonatal death rates (especially the former) increase when pregnancy goes past term (Clayton, 1941, 1953; McKiddie, 1949). Analysis showed that the rise in the obstetric death rate in post-maturity could be attributed to two groups of deaths, "mature, cause unknown" and "trauma." The association of these two groups is not unexpected, since if a baby is liable to die without obvious cause it is presumably all the more likely to die if the stress of prolonged or difficult labour is added. Furthermore, it is known that the incidence of difficult labour increases after term.

Analysis of the incidence of foetal distress in labour showed that it behaves similarly to the "mature, cause unknown" death rate, increasing rapidly with maternal age in primigravidae and in post-maturity.

On the basis of these facts an attempt is being made to reduce the incidence of "mature, cause unknown" deaths by (*a*) routine induction of labour at the end of the forty-first week in all post-mature primigravidae over 25 years of age, and (*b*) more frequent resort to caesarean section when foetal distress, especially the passage of meconium, is noted during labour. Results to date are encouraging. Epidemiological research has thus pointed the way to a positive method of saving lives when the real cause of death is unknown.

It may be noted in passing that this kind of epidemiological research is possible only if very detailed clinical

information is available, and therefore ought to be undertaken primarily by clinicians rather than by statisticians. Some knowledge of statistical methods is useful, but it is much more important to tabulate accurate information sensibly and to interpret trends with insight than to calculate precisely the vagaries of chance among data which may be neither accurate nor meaningful.

Impaired Foetal Oxygenation

Finally, what is the cause of "mature, cause unknown" death and of foetal distress during labour? The association with post-maturity and the fact that anoxia is a common finding at necropsy suggest that impaired oxygenation of the post-mature foetus may be the immediate cause. This is supported by the work of Barcroft and Young (1945) on animals. Walker (1954) has undertaken a study of the oxygen content of human foetal blood and has found that the percentage saturation—that is, the content in relation to the capacity—falls after term in normal pregnancy, and is unusually low where there is foetal distress and in a second labour in cases in which the first pregnancy ended in a "mature, cause unknown" death. These findings confirm the hypothesis that impaired foetal oxygenation is the main reason for the excess foetal mortality after term; presumably the basic reason has to do with a decline in the functional efficiency of the placenta when pregnancy is unduly prolonged. We have yet to determine whether the high obstetric death rate in elderly primiparae is due to exceptionally low foetal blood-oxygen levels or to normal levels together with foetuses of low general vitality; it is not due to an unusually high incidence of post-maturity.

Summary and Conclusions

The study of morbidity and mortality statistics requires detailed clinical knowledge and good clinical case records if it is to be most fruitful. Statistics of maternity present many natural advantages for such a study, which should preferably be based on an administrative area. The scientific value of the traditional annual reports compiled by maternity hospitals is extremely limited.

The epidemiological approach in obstetrics is useful in providing insight into the nature and means of prevention of conditions which cannot be fully understood by detailed study of the individual case. Besides providing perspective, epidemiological research in a clinical unit increases precision of clinical observation, improves the quality of case records, suggests fruitful lines of laboratory investigation, and provides the means of checking the results of treatment.

The approach is illustrated by researches into the nature of unexplained stillbirths and first-week deaths in Aberdeen.

I am indebted to Professor Dugald Baird, Dr. James Walker, and other colleagues for valuable help and for allowing me to report these results of team research; to members of the Medical Research Council's Social Medicine Research Unit (Director, Dr. J. N. Morris); and to the Advisory Committee on Medical Research (Scotland).

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THE TREATMENT OF SUBACUTE AND CHRONIC SUBDURAL HAEMATOMAS

BY

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Wilfred Trotter (1914) recognized that some of the poor results in treating chronic subdural haematomas were due to failure of the brain to expand after their evacuation. This observation has been confirmed many times. The head-down position, intravenous fluids, and drainage of the subdural space have been used to help the brain to resume its volume after removal of the compressing mass. LaLonde and Gardner (1948) showed that better results were got when saline was injected into the subarachnoid space to help the brain to expand. Since then no other series of cases so treated has been reported.

Details of Treatment

It is common practice to seek and treat subdural haematomas by making burr-holes in the skull. There is no value in raising an osteoplastic flap, and it may be detrimental. Improved methods of diagnosis have precluded cases in which a subdural haematoma was a surprise finding at craniotomy for a cerebral tumour.

The making of burr-holes is always done under local analgesia, no matter how restless the patient. The usual site for the initial burr-hole is in the mid-temporal region about 1½ in. (4 cm.) above the zygoma. Further burr-holes may be made in the frontal and parietal areas. The most useful site for the second burr-hole is in the posterior parietal region. While a fluid haematoma can be easily treated through one burr-hole, a clotted haematoma needs multiple explorations. Bilateral burr-holes are always made, as the haematomas are bilateral in many cases. Incision of the dura mater will allow the haematoma contents to gush out. A fine rubber catheter is introduced into the subdural space in all directions and irrigation is carried out gently with Ringer's solution, which is preferable to normal saline. The washing is continued until the returning fluid is clear. In favourable cases the brain will expand to fill the dead space left by evacuation of the haematoma. The skin incision is closed in two layers and no drainage tubes are inserted.

The intrathecal injection of Ringer's solution is used in every case in which the brain does not expand after removing the haematoma. Ringer's solution can be injected into the lumbar subarachnoid space or into the ventricles of the brain by way of one of the burr-holes. Ventricular injection may be difficult if the ventricles are collapsed, and some experience is needed for ventricular puncture. Lumbar puncture is easier, and is safe and effective. When the haematoma is unilateral the towelled patient is not greatly disturbed on being turned from the supine to the lateral position for lumbar puncture. It is unsatisfactory, however, to turn patients with bilateral subdural haematomas. This difficulty has been overcome by operating with the patient in the prone position. The head is held in a cerebellar head-rest, which is adjusted to the patient's comfort, no flexion of the head being needed. No respiratory embarrassment occurs in this posture if the chest is raised on the shoulder-pieces, as the tongue cannot fall back into the pharynx. The lumbar-puncture needle may be inserted before the burr-holes are made. The prone position gives good access to all parts of the head except the forehead. It is satisfactory also for making burr-holes in cases of acute head injury. The patient is in an ideal position if the posterior fossa has to be explored for a haematoma.

Between 50 and 200 ml. of Ringer's solution will be needed for brain expansion. The injection should be continued until the brain is in contact with the edge of the burr-hole. A recent case of bilateral subdural haematomas of infancy had 160 ml. of fluid injected by lumbar puncture without any adverse effect. When a single burr-hole is used as a means of treatment it is better to leave the catheter as a vent over the convexity of the brain until expansion has removed surplus fluid and air from the subdural space.

The after-treatment consists in keeping the patient flat and maintaining a good fluid intake. When the inflation method has been used it is desirable to do a daily lumbar puncture for at least four days. If the cerebrospinal fluid pressure is below 150 mm. H_2O Ringer's solution is injected to raise the pressure to this level. The state of consciousness and the absence of headache are reliable guides to clinical improvement.

Clinical Material

Twenty-eight cases of subacute and chronic subdural haematomas have been treated in the neurosurgical unit at Dunedin Hospital. Subdural haematomas of infancy and one case of chronic subdural haematoma in a child of 5 years with a successful outcome are excluded from the series. The subacute haematomas occurred between seven and twenty-one days of a known head injury. The chronic haematomas had a latent period of over three weeks, or a history of previous injury was unobtainable. The ages of the patients ranged from 17 to 77 years.

No fresh observations can be added to the well-known clinical picture. Many patients were seriously ill and had come long distances—often up to 500 miles by air ambulance. Eighteen had had lumbar puncture, mostly in other hospitals, and eight were made much worse by this procedure. In most cases the information gained by lumbar puncture did not materially assist in the diagnosis, and its danger in these cases cannot be too greatly stressed.

The diagnostic procedure of choice was carotid arteriography. When an arteriogram indicated a haematoma, an examination of the other side was not inflicted upon the patient, and a subsequent burr-hole was used instead. If the previous head injury was clear-cut or the condition of the patient with suspected haematoma was parlous, immediate burr-holes were preferred to any other diagnostic method. We have been reluctant to use arteriography in patients over the age of 65, and have not used it at all in those over 70. At this age there are few alternatives in the diagnosis of subdural haematoma, and the making of burr-holes is a benign matter.

In 1952 it was decided that the inflation method should be used whenever the brain did not expand after removal of the haematoma. Ten patients come under the old regime and 18 are in the new group. In 14 of the latter inflation was used. The subdivisions showed the same degree of neurological severity. However, there were four patients over 70 years and three with bilateral haematomas in the later group. This 10% incidence of bilaterality is at variance with the 30% incidence in most other series.

There was one death, and that was in the first group. The patient was a semi-comatose man who died 24 hours after burr-holes had been made. The necropsy showed that the brain had not expanded. Inflation might well have saved him.

The outstanding difference between the two groups was in the degree of recovery. Under the old regime, patients often remained confused and had headaches for several days—sometimes a week or more—and during that time gave cause for concern. There was one recurrence, and that was dealt with successfully. The new method brought a more rapid return to full consciousness. This improvement was often apparent on the operating table, and the post-operative period was less worrying. It was common for the headaches to disappear from the time of inflation. The greatest advantage was in elderly patients, who seem to be prone to develop dehydration and intracranial hypotension. While it is impossible to be dogmatic, it is felt that a fatal issue was averted in several cases by the use of inflation. No ill effects from this treatment were seen.

When inflation was first used it was hoped that a single injection would be enough and that recurrences would be avoided. There were two patients over 70 years who made a dramatic improvement for a few days and then suddenly relapsed: further collections of fluid were successfully treated. Experience has shown that the only way to prevent recurrences is to do a daily lumbar puncture and to keep the C.S.F. pressure at 150 mm. H_2O by further injection as recommended by Munro (1952). The minimum period for this procedure is four days, and it must be continued until the patient's condition has stabilized.

Six patients had their inflation done by lumbar puncture and six had direct ventricular injection. The other two had combined lumbar and ventricular puncture. There is nothing to choose between the results of each method.

Discussion

The conclusions of LaLonde and Gardner (1948) on the value of injection of normal saline into the subarachnoid space or ventricles in the treatment of subdural haematomas are confirmed; but they did not give their results before using the method. Munro (1952) recommends the same treatment for all types of subdural haematoma. His use of repeated lumbar puncture to sustain the C.S.F. pressure is essential if recurrences are to be prevented.

The critical features in the train of events initiated by a subdural haematoma are compression of the brain, tentorial herniation by the temporal lobe of the brain, and intracranial hypotension. Jefferson (1938, 1951) has given good reviews of tentorial herniation, and intracranial hypotension has been reviewed by Page (1953) and Holmes (1953). In many cases removal of the haematoma leads to immediate expansion of the brain and rectification of these three complications; but they may persist singly or in combination and be responsible for a slow recovery or a fatal issue. The method of expanding the subarachnoid space and the cerebral ventricles, and also the brain, will counteract these adverse features. So long as there are no irreversible changes, the mortality should be lower and convalescence more rapid than with mere evacuation of the haematoma. This series, however, is too small for dogmatism regarding the mortality, though there is no doubt about the improvement which occurred in the recovery period when the injection method was used.

The method of brain expansion, whether ventricular or lumbar, does not make any difference to the end-result. The lumbar route is the easiest for the less experienced. The use of the prone position for the patient is an advantage for lumbar puncture and for access to the head.

Summary

The treatment of subacute and chronic subdural haematomas by the injection of Ringer's solution into the lumbar theca or cerebral ventricles is advocated.

Comparison is made between 10 cases before and 18 cases after this method was used.

Some of the pathological features of subdural haematoma are discussed in relation to treatment.

I wish to thank Mr A. James for permission to use the cases admitted under his care and for access to the departmental records. He has given every encouragement in treating some of these cases and in writing this paper.

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SPONTANEOUS RUPTURE OF OESOPHAGUS

REPORT OF THREE SUCCESSFUL CASES TREATED SURGICALLY

BY

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Spontaneous rupture of the oesophagus, until 1947 a fatal disease, can now be effectively treated by surgical means. The criterion for success requires that such a lesion be known about and kept in mind, that it may be recognized early and treated promptly. The general practitioner is as able to make the diagnosis as the specialist; and, like perforated duodenal ulcer, early recognition and treatment gives satisfying results (Borrie, 1953).

This paper reports three cases seen within an eight-months period at one Thoracic Surgical Unit dealing with a regional area of 250,000 people. In none of the three cases was the diagnosis at first suspected; in all, however, it was later established and operation was followed by complete recovery.

Case 1

A married woman aged 65 was an emergency admission to the medical wards of Dunedin Hospital on December 15, 1952, under Professor J. E. Caughey, with a diagnosis of haematemesis. Earlier that evening, while hungrily eating a fish meal, she tried to make herself sick because she thought she had swallowed a fishbone. She vomited copiously 1 pint (570 ml.) of bright blood-stained gastric contents, and was immediately seized with severe chest pain.

On examination she complained of pain behind the lower third of the sternum. It was severe and gripping, and came in spasms. There were no abnormal respiratory, cardiovascular, or abdominal physical signs, and no cervical emphysema. Her blood pressure was 180/110 mm. Hg, and her haemoglobin 90% (Sahli).

A diagnosis of haematemesis secondary to a foreign body in the lower third of the oesophagus was made, and she was referred to the ear, nose, and throat department. It was decided to keep her under close observation, with nothing by mouth, a rectal drip, penicillin, and "omnopon" to relieve her pain.

On the morning of December 16 she had ceased vomiting, looked better, but still had retrosternal pain. Later that day she could not swallow her sputum, and regurgitated blood-stained mucus. Oesophagoscopy was performed that evening by Mr. Alan Wardale, surgeon in charge of the ear, nose, and throat department. He found a considerable amount of dark blood lying in the oesophagus. The upper two-thirds of the oesophageal mucosa in particular was haemorrhagic. There was no foreign body. The possibility of rupture of the oesophagus at the junction of the middle and lower thirds was entertained, and on December 17 she was referred to the thoracic surgical department.

Clinically there was still no pleural effusion or cervical surgical emphysema. Radiopaque oil was given, and fluoroscopy showed a vertical rent on the left side of the lower third of the oesophagus, lying 1 in. (2.5 cm.) above the diaphragm. The diagnosis of spontaneous rupture of the oesophagus was established and left thoracotomy advised.

Operation.—Pre-operative oesophagoscopy confirmed haemorrhagic oedema of the entire oesophageal mucous membrane together with a vertical ragged area in the lower third of the oesophagus. Under general anaesthesia, the left chest was opened through the bed of the resected left eighth rib. There was an extensive extravasation of blood-stained fluid under the mediastinal pleura and extending from the level of the oesophageal hiatus in the diaphragm up to the left inferior pulmonary vein, and midway between the necks and angles of the lower left ribs. Through a 3-in. (7.5-cm.) incision in the mediastinal pleura, the oesophagus was mobilized and retracted with a Jaques rubber catheter sling. On its left border was the vertical tear, approximately ¾ in. (1.9 cm.) in length. The points of dissecting scissors were easily introduced through the tear into the oesophageal lumen. The tear was closed with four interrupted twenty-day No. 1 chromic catgut sutures passed through all layers. The mediastinal pleura was not sutured. The left chest was closed over water-seal drainage, using interrupted catgut for the muscle layers and fine nylon for the skin.

The patient was allowed sips of water by mouth and over a period of three days gradually increased her intake by mouth to a fluid diet. She took solid food a week after operation. The intravenous fluid was discontinued on the third post-operative day, and the intercostal drainage was removed on the fifth post-operative day.

She was discharged, convalescent, three weeks after admission, swallowing normally. Subsequent fluoroscopy and oesophagoscopy showed no abnormality of the foregut. When seen 18 months after operation she remained well.

Comment.—This patient illustrates the typical onset following violent vomiting, and shows how a small tear need not be associated with rupture into the pleural cavities or with detectable emphysema. The constant retrosternal pain following vomiting alone suggests the diagnosis, which is further suspected after unusual findings at oesophagoscopy, and convincingly established by careful fluoroscopy with radiopaque oil. Untreated, she would undoubtedly have developed a serious mediastinal abscess.

Case 2

A bachelor aged 53 was brought to the casualty department of the Dunedin Hospital at 9 p.m. on June 29, 1953. Earlier that day he was well, and between 5 and 6 p.m. drank six pints (3.4 litres) of beer in a hotel. When the bar officially closed at 6 p.m. he staggered into the street and vomited copiously into the gutter. He started to walk away, but had just gone five paces when he suffered an agonizing retrosternal pain which radiated to the left shoulder and leg. He drove his car home with great difficulty, the pain becoming more intense and breathlessness more acute. There was no further vomiting.

When examined on admission he still had excruciating pain, was restless, and quite unable to find relief either by sitting up or lying down. He was convinced he would die. His pulse was 120 and blood pressure 170/110 mm. Hg. The casualty officer found the apex beat normal. What was believed to be a pericardial friction rub, crackling in quality, was detected. There were left basal crepitations. The abdomen was board-like, but with no definite tenderness. Although acute pancreatitis or rupture of the gall-bladder was entertained, these diagnoses were not supported by the general surgeon who was called to see him.

An E.C.G. showed T-wave inversion, with tall R waves in V_5 strongly suggesting left ventricular strain. With the diagnosis still in doubt, a chest radiograph was ordered. At this stage surgical emphysema above the suprasternal notch was noted, the correct diagnosis established, and the patient admitted to the thoracic surgical department. The chest radiograph showed left hydropneumothorax with air around the aortic arch and soft tissues of each side of the neck.

Operation.—At midnight, under general anaesthesia, the left chest was opened through the left ninth rib bed. The pleural cavity contained 1½ pints (850 ml.) of brown alkaline fluid. The mediastinum was emphysematous and the lacerated pleura had lifted from off the aorta and oesophagus. A catheter sling was passed around the oesophagus,

which was then lifted up for inspection. On its left side was a vertical rent 1 in. (2.5 cm.) long, and starting 1 in. (2.5 cm.) above the oesophageal hiatus in the diaphragm. Through this rent protruded oesophageal mucosa. After inserting mucosal stay sutures, the tear was closed with a series of interrupted chromic catgut sutures passed through all oesophageal layers. The chest wall was closed as in Case 1.

On July 1, because of sputum retention, aspiration bronchoscopy was performed and gave relief. The intercostal drainage was removed on the third post-operative day, and the skin sutures on the seventh day. The patient was discharged fit three and a half weeks after admission. The post-operative feeding regime was the same as in Case 1.

Subsequent barium study of the foregut revealed no pleural or oesophageal abnormality, but suggested a deformity of the duodenal cap without any definite evidence of active ulcer. Eleven months after operation the patient continued well and was working.

Comment.—This case illustrates the classical onset of symptoms first graphically described by Boerhaave in 1723 (Barrett, 1946). It illustrates their confusing severity, and how tell-tale cervical emphysema gave the key to diagnosis. Physical findings in the chest had been difficult to elicit and interpret, and an E.C.G. record was definitely misleading. It further shows how prompt surgical intervention materially altered the course of a catastrophe, with minimal convalescence and a most gratifying result.

Case 3

A farmer aged 52 was admitted as an emergency case to a county town hospital on July 20, 1953, suffering from severe epigastric and right chest pain. For years he had had an active duodenal ulcer; for four days before admission there had been an acute exacerbation of his ulcer symptoms, and he took only milk diet until 7 p.m. on July 20, when he ate a pear. Half an hour later he vomited twice and suddenly developed severe pain in the right chest, shoulder, and upper abdomen. The vomit was "coffee ground" material.

On admission he was distressed, with grunting respirations and slight cyanosis; pulse 120 and blood pressure 140/90 mm. Hg. The tongue was dry and furred. There was generalized abdominal rigidity, and absent bowel sounds. Examination of the chest at that time revealed no obvious abnormalities. A diagnosis of perforated duodenal ulcer was logically made, and the patient was prepared for operation—a Ryle tube being passed into the stomach and aspirated, and an intravenous drip being started.

First Operation.—At 11.30 p.m. that same night, under general anaesthesia with endotracheal intubation, laparotomy through an upper right paramedian incision showed the presence of a chronic duodenal ulcer in the first part of the duodenum and firmly adherent to the pancreas. There was no perforation, and the pylorus was patent. The large dilated stomach was further aspirated, 42 oz. (1,200 ml.) of brown fluid being removed. The abdomen was closed and the patient returned to the ward.

On the morning of July 21 a chest radiograph revealed a right-sided hydropneumothorax without tension phenomena. Spontaneous rupture of the oesophagus was established. Throughout the morning his condition gradually deteriorated, nor was it improved by aspirating a few millilitres of that same brownish fluid from the right pleural cavity. His pulse was 140 and blood pressure 55 mm. Hg. Plasma infusion, oxygen, and penicillin were given with no great improvement.

After telephone consultation with the thoracic surgeon in Dunedin, a Malécot catheter was inserted into the right pleural cavity and connected to water-seal drainage. His blood pressure steadily rose to 90 mm. Hg. A pint (570 ml.) blood transfusion was started and the patient was transferred 70 miles by ambulance.

At 11 p.m. on July 21 he was admitted to Dunedin Hospital, having withstood the journey well. Both the blood drip and the intercostal drainage were working satisfactorily. His temperature was 100.5° F. (38° C.), pulse 114, respirations 48, and blood pressure 110 mm. Hg systolic. A further chest radiograph showed little alteration, and the right lung was still partially collapsed.

Second Operation.—An hour later general anaesthesia was induced and the right chest opened through the ninth intercostal space. The pleural cavity contained ½ pint (280 ml.) of brownish fluid. With positive pressure, the right lung was made to re-expand. Below the azygos arch was an irregular tear in the mediastinal pleura, with surrounding oedema. The oesophagus was isolated from the mediastinum and a catheter sling passed round it. Inspection revealed a vertical rent 1½ in. (3.8 cm.) long, starting 2 in. (5 cm.) above the diaphragm, and extending upwards to the azygos arch. This was closed with a series of No. 4 silk sutures, placed ¼ in. (3 mm.) apart and passed, as before, through all oesophageal layers. The chest was closed over water-seal drainage.

Post-operatively, bronchoscopy was performed and he was returned to the ward, where graduated continuous suction was applied to the water-seal drain. Fluids by mouth were restricted for four days, during which time intravenous therapy was continued. He was given penicillin and streptomycin.

A chest radiograph on July 24 confirmed that the right lung had re-expanded and was aerating satisfactorily. On July 27, however, there was evidence of a loculated basal pleural effusion, and on July 31 this was drained. The presence of a fistula and also staining after swallowing methylene blue solution confirmed that there was a leak from the oesophageal suture line.

On August 1 a second anterior pocket of pus in the right pleural cavity was cleared by intercostal drainage, and a Stamm gastrostomy performed. Progress thereafter was slow. The partially collapsed lung required a third apical intercostal drain and continuous suction for two weeks before it had re-expanded.

By September 30, almost two months after admission, the oesophageal fistula had healed and normal swallowing was again allowed.

On October 8 the gastrostomy tube was removed, by November 5 the anterior pleural sinus was healed, and by November 18, as the posterior sinus was negligible, he was discharged. He was last seen on March 10, 1954, when his sinuses were well healed and his swallowing was satisfactory.

Comment.—This is an interesting case of cause and effect. It demonstrates a further and unusual complication of duodenal ulcer—a complication which must always be kept in mind when sudden and severe abdominal symptoms follow vomiting. One can readily appreciate how the rupturing of the oesophagus into the right pleural cavity, with associated epigastric pain and rigidity, led to the possible diagnosis of perforated duodenal ulcer. Undoubtedly a pre-operative chest radiograph would have revealed the true diagnosis. This case, and two others not yet in the literature but known by me to have been explored as acute pancreatitis, induce me to plead for a pre-operative chest radiograph in all difficult acute abdominal cases. Nowadays, in such circumstances, a preliminary abdominal radiograph is common practice. The same film can well be adjusted to include the diaphragm and the lower chest, and thus avoid needless laparotomy. The case further illustrates how a patient in civilian practice, with pneumothorax and empyema made safe and temporarily relieved by intercostal drainage and by blood transfusion, can travel 70 miles by ambulance and be improved at the end of his journey.

Although Susman (1953) wisely points out that some cases of spontaneous rupture can recover without suture of the rent, and that Barrett's (1948) case of suture in fact leaked after surgical closure to produce a further oesophageal fistula and empyema, yet such conservative measures must

include adequate treating of the accompanying empyema. In a late case, with fully established empyema, to attempt to free the oesophagus and perform suture would be undoubted folly. In an early case, however, possibly up to 48 hours after perforation, the right attitude should be to explore and if possible suture the rent.

In this third case—with a long rent—there was a late leak. By performing a gastrostomy as an oesophageal bypass, and thus restricting swallowing purely to saliva, local pleural drainage together with gastrostomy ultimately gave sound closure.

Summary and Conclusions

Spontaneous perforation of the oesophagus is an acute emergency which must always be kept in mind and which may simulate many lesions. It can masquerade as coronary thrombosis, or acute abdominal catastrophes, especially perforated gastric ulcer, acute pancreatitis, and acute cholecystitis. Frequently the patient is too distressed to give a rational history.

As the oesophagus may perforate into the mediastinum alone, into the right or left pleural cavities, or into both, there is a diversity of signs and symptoms. Convincing chest clinical signs are often hard to elicit. Not all cases have cervical emphysema, but, if present, it is diagnostic. Chest radiographs, including, if necessary, the swallowing of radiopaque oil, confirm the diagnosis.

Pre-operative management embodies prompt relief of any tension pneumothorax, restriction of all food by mouth, intravenous fluids, passage of a Ryle tube to the stomach, and antibiotics.

Thoracotomy is then required, and wherever possible the rent should be sutured. In early cases, healing is sound and convalescence greatly shortened.

A plea is made to include a chest radiograph when faced with a difficult diagnosis of the acute abdomen.

I gratefully acknowledge help and co-operation with these patients from Professor J. E. Caughey, Mr. Alan Wardale, Mr. C. L. Tucker, Dr. R. L. Coulter, Dr. T. O'Donnell, and my resident staff in the Dunedin Hospital. I also wish to thank Dr. James Thomson, acting medical superintendent of the Dunedin Hospital, for permission to publish these records.

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An observing station for measuring the smoke and sulphur dioxide pollution in the London atmosphere has been established in the Science Museum by co-operation with the Fuel Research Station of the Department of Scientific and Industrial Research. The station is one of a number that are being established to permit a more detailed examination of atmospheric pollution in London and elsewhere. The sampling equipment is displayed as a working exhibit in the Meteorology gallery. The smoke particles are captured on filter paper through which air is drawn from just above museum roof level by means of a small electric pump. As a result a grey stain appears on the filter paper, the intensity of which is a measure of the degree of smoke pollution. The same air is bubbled through a weak solution of hydrogen peroxide, where the atmospheric sulphur dioxide is dissolved to form an acid solution, the strength of which is measured by standard chemical methods. Observations of smoke and sulphur dioxide pollution are made daily by a member of the museums staff and reported to the Fuel Research Station, who are in charge of the investigation.

SPONTANEOUS RUPTURE OF OESOPHAGUS

BY

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The following case of spontaneous rupture of the oesophagus is placed on record, even though treatment was unsuccessful, as this condition is still thought to be rather rare. The records of this hospital for the past six years, covering 28,725 admissions, show only one comparable case—that of an eclamptic patient who died thirty-two hours after admission—in which two perforations involving the lower end of the oesophagus were found; unfortunately the data about this case are too scanty for an accurate diagnosis to be made.

Increasing awareness of the condition seems to be bringing more cases to light. Mallam, Whitelocke, and Robb-Smith (1940) stated that the total number of published cases was 40. Barrett (1946), in a comprehensive review of the subject, put the figure at about 50 cases. Mauro (1952) discussed 103 cases in the literature. Barrett (1947) was the first to describe recovery following correct diagnosis and deliberate operation to repair the rupture. Frink (1947) published details of a successfully treated case of 1941, and Samson (1951) recorded two such cases mentioned by Graham in 1944. Nanson and Milnes Walker (1953) added the twenty-third recovery to the literature.

It would be wrong to publish only the successes in the treatment of this condition if a correct record of mortality rate is the aim. The mortality rate may be even higher and the disease commoner than the records would suggest, as up to 1949 only 16 cases had been diagnosed in life and the remainder at necropsy (Russell, 1953), and it may be possible to miss a ruptured oesophagus at necropsy unless one is looking especially for the condition (Barrett, 1946). Many cases of death in a period of hours to a few days after the onset of retrosternal pain, collapse, and perhaps vomiting must have been certified as being due to coronary thrombosis without necropsy confirmation, and these might well include a number of cases of spontaneous rupture of the oesophagus.

Summary of Case Notes

A 73-year-old widow, about to begin her tea, complained of severe upper abdominal pain which rapidly spread to both sides of the lower chest and back. After 30 minutes she vomited a little blood-stained fluid and then collapsed with pain. She was admitted to the medical wards, and after several hours was transferred to the surgical side. A long history of dyspepsia which she relieved by making herself vomit was obtained.

Examination.—Grossly obese, very shocked, and in great pain. Respirations 24, pulse 80, temperature taken rectally 99.8° F. (37.7° C.); blood pressure 150/90 mm. Hg. Tongue stained with altered blood. Marked surgical emphysema of front of neck. Equal aeration of both lungs and no detectable effusion or pneumothorax. Abdomen very fat, with large irreducible umbilical hernia; tender in right epigastrium but no guarding. A Ryle tube was passed and 90 ml. of thick bloody material was aspirated.

A provisional diagnosis of spontaneous rupture of the oesophagus was made and the patient was transferred to the *x*-ray department. Films of the chest were taken and then three mouthfuls of barium mixture were given to her to swallow before repeating the pictures. The following

information was obtained from these. The Ryle tube was folded on itself in the oesophagus. There was marked widening of the mediastinum and much free gas was present in it. There was slight collapse of the medial lung substance at both bases and a very little free pleural effusion on each side, but no pneumothorax. Barium was found to have passed into the posterior mediastinum to the left at the level of the lower part of the body of the seventh thoracic vertebra (see Fig.).

Radiograph showing barium in posterior mediastinum.

Operation. — In view of the certainty of a fatal outcome if untreated, operation was undertaken in spite of the patient's age and poor physical state. Through a right thoracotomy, the intact mediastinum was widely opened, releasing air under pressure, blood, and pieces of chicken meat (partaken of four hours before the onset of the condition). The tear was not located, but the left side of the lower oesophagus could not be seen satisfactorily from this approach. The search was not prolonged, and a wide-bore stomach tube was passed without revealing the tear. The chest was closed after inflating the right lung, and two intercostal drainage tubes, leading from the mediastinum and the pleural cavity to underwater seals, were inserted.

After 36 hours her condition deteriorated and she died 48 hours after operation.

Post-mortem Examination.—The right lung was fully aerated and adhering to the parietal pleura; the mediastinum was dry and relatively clean. The left pleural cavity contained 2 pints (1,140 ml.) of clear brownish inflammatory exudate, but the mediastinal pleura was intact. The oesophagus had a vertical tear 2 in. (5 cm.) long on its left posterior aspect, the lowest part of the tear being 1¼ in. (3 cm.) from the oesophago-cardial junction. The myocardium was pale and fatty and the right side of the heart was dilated; other organs showed evidence of congestive failure. Microscopical examination of many sections of oesophagus about the tear showed no evidence of pre-existing disease and the appearances were consistent with spontaneous rupture.

Discussion

Mackler (1952) distinguishes between rupture and perforation of the oesophagus, the term "rupture" applying when it occurs in a previously normal oesophagus and is not caused by direct trauma. Spontaneous rupture is usually associated with vomiting, but occasionally with other causes of increased intra-abdominal pressure. Mackler experimented with 65 oesophagi from fresh cadavers, by inflating them with air, and in all but three cases rupture took place as a longitudinal tear at the lower end of the oesophagus in its left lateral wall. McKenzie (1884) had also demonstrated this.

In the present case the tear was typical, as were the symptoms. The pleural cavity was not directly involved, as it often is (usually on the left side). No history of vomiting prior to the rupture could be obtained, though there was a long history of habitual voluntary vomiting.

Recent writers mention the value of radiography in the diagnosis. Dorsey (1951) states that the patient should be given radiopaque material to swallow, and if none escapes from the oesophageal lumen no harm will follow; if it does escape, operation is indicated. Nanson and Milnes Walker (1953) used a barium compound, screening their patient; provided surgery follows if indicated, there can be no danger in using barium mixture. Priviteri and Gay (1951) prefer lipiodol to demonstrate the fistula, its site and extent.

In the above case barium mixture was used. It escaped only from the uppermost part of the tear, giving the impression that the tear was perhaps smaller and higher than it was eventually found to be.

Most writers are agreed that early diagnosis and immediate operation are necessary if there is to be recovery. Operation and drainage of the mediastinum, with the rupture of the oesophagus being adequately by-passed for some time, has been successful in a number of cases, and Jelliffe and Ferguson (1953) suggest that it may be sufficient. But most authors appear to feel that repair of the oesophageal defect increases the patient's chances of recovery. Mackler (1952) assesses the percentage of recoveries after drainage alone at 33%, and after definitive repair of the oesophagus at 69%.

Operation should preferably be performed at a centre with experience of operating in this field, but, as in the above case, the patient may not be fit to be transported any distance.

Summary

A case of spontaneous rupture of the oesophagus is briefly described.

The value of radiography and the use of radiopaque media as a swallow, to establish or confirm the diagnosis, is affirmed.

I should like to express my appreciation to Mr. L. Wynn Houghton for his advice and assistance with this case, and his permission to publish it.

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A new British–American hospital, established to serve the constantly growing needs of the English-speaking community, has been opened in Madrid by Sir IVO MALLET, the British Ambassador. The opening ceremony, attended by representatives of the Spanish Government and British and American residents, marked the end of three years' work to provide more adequate accommodation than that available in the 5-bed British–American nursing-home opened in 1923. Built on rising ground in the faculty of medicine area of Madrid's University City, the new 21-bed hospital stands on land leased free of charge for 50 years by the university authorities. A long, low, red-brick building on three floors, so constructed that three more floors can be added to meet future needs, it has a large garden and a wide view across the University City to the snow-capped peaks of the Guadarrama mountains north of Madrid. Several American firms gave medical and surgical equipment or sold it at cost, and an iron lung, said to be the only one in Spain for civilian use, was given by the U.S. Public Health Service. Equipment bought in England was shipped free of charge by a British mining company operating in Spain. The idea of building a new hospital for the British–American colony was proposed in June, 1951, by Sir John Balfour, then British Ambassador, in collaboration with Mr. Stanton Griffis, an American businessman, who at that time was U.S. Ambassador to Spain.

BRUCELLOSIS AS A HAZARD OF BLOOD TRANSFUSION

BY

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Since the introduction of low-temperature banks for the storage of blood, transmission of disease by transfusion has become negligible; Mollison (1951) and Whitby and Britton (1953) quote only syphilis, malaria, hepatitis, and some virus diseases as a possibility. Meno Brito (1948) refers to two cases reported at the second Mexican Brucellosis Congress in 1944, in which brucellosis was clearly transmitted by blood transfusion. Subsequently, during two years' work at a children's hospital he found five more instances in which brucella was isolated from the blood of donors, with no indication of clinical brucellosis, but whose blood on transfusion gave rise to the disease in recipients. He gives a table, showing the percentage of positive agglutinations against *Br. abortus* (1.31% in 2,899 donors) recorded at the hospital in 1945 and 1946, and states that, although a positive reaction is not sufficient for the diagnosis of active infection, he recommends that, in Mexico, tests should regularly be carried out to exclude the presence of agglutinins in the donor's blood. Brucellosis has a much lower incidence in this country, but the occurrence of the case described below shows the importance of questioning prospective donors and rejecting those whose state of health is unsatisfactory, particularly those who may be in the incubation stage of an infective disease.

History of the Donor

A married woman aged 53 was a regular donor who had never before felt any after-effects, but who returned home from her donation on October 11, 1952, feeling "very unwell." She did not consult her doctor for 10 days, but complained afterwards that she had been ailing all this time, and a fortnight later, on November 5, she was admitted to Southmead Hospital, Bristol, with undulant fever. On November 7 her serum agglutinated *Br. abortus* to a titre of 1:1280. A blood culture taken at the same time was sterile after 14 days' incubation.

In view of the history and the diagnostic level of the titre four weeks after donation, it seems likely that this woman's blood would have shown a bacteriaemia at that time.

History of the Recipient

The recipient, a man aged 27, had lived a "transfusion life" since 1946, when he developed a complete red-cell aplasia shortly after painting with "aeroplane dope." On October 17, 1952, he received his usual weekly transfusion of one pint (570 ml.) of blood, and early in November a report was received from the Regional Transfusion Centre that the donor was suffering from brucellosis. Questioned at his next visit, the patient admitted that he had had a cold, but his temperature was normal and his serum agglutination tests were negative, as they were on three subsequent occasions up to December 28. Indirect questioning failed to elicit any other complaint, and it was not until January 17, 1953 (13 weeks after the transfusion), that he admitted feeling unwell; he then complained of malaise, worse in the morning, slight sweating, also more pronounced in the morning, anorexia, some diarrhoea, and vague pains in the limbs. His temperature was 99° F. (37.2° C.), and examination showed a purpuric rash on both limbs; the liver and spleen were just palpable. His liver function was known to have been depressed since a homologous serum jaundice in 1949, and there was a severe transfusional haemosiderosis. On January 18 his serum agglutinated *Br. abortus* to a titre of 1:2,560; he was treated with chlortetracycline ("aureomycin"), 250 mg. six-hourly for six days, and his temperature fell to normal. He remained depressed, however, and failed afterwards to attend regularly for transfusions.

He was unable, or unwilling, to give an account of the beginning of his illness, and a visit was paid to his home in the hope of obtaining some information. His mother remembered that he first complained of nausea and abdominal discomfort towards the end of October, when he thought "the jaundice was coming back"; she bought him some medicine from a chemist but did not consult a doctor. Jaundice did not, however, develop. It was decided that he had "gastric 'flu," and he was in bed, on and off, for several weeks. By Christmas he was very unwell, eating little and feeling increasingly depressed, but by the middle of January his illness was, in his mother's opinion, abating. His milk-drinking habits were investigated, and it seemed certain he took very little milk, and that scalded, in tea and milk puddings. A report from Mr. H. G. L. Harvey states that in his opinion the weight of evidence points to the absence of brucellar abortion on the farm supplying the milk; unfortunately only one of the two cows owned at the time could be traced, but tests on this animal were negative.

Discussion

Spink and Anderson (1950) discuss the transmission of brucella through the blood of an asymptomatic but bacteriaemic individual; they believe that, although a scanty inoculum of brucella will remain viable up to six months at 4° C., from a practical point of view the likelihood of transmitting disease by this route is remote because the organism would be destroyed by the blood or tissues of the recipients. That patients with active disease have relatively few organisms is shown by the difficulty of blood culture and the few colonies generally obtained.

In the above case the recipient had been suffering from a severe aregenerative anaemia and was dependent on transfused red cells for his continued existence. His reticulo-endothelial system was known to be damaged, and it is tempting to speculate that as a result of this he was less competent than a normal individual to deal with the small number of organisms transfused. Agglutinins in this man developed late, but ultimately were in high titre, and it is recognized that a delay of weeks or months may occur even in the normal individual—indeed, that the whole process may pass without any detectable agglutinins being formed at all (Evans, 1934). Hardy *et al.* (1938), studying cases diagnosed in New York whose onset can be accurately dated because the entire milk supply to the city is pasteurized and infection by this means can result only from visits outside the Metropolitan area, stress the insidiousness of onset of the disease and the uncertainty regarding the early symptoms in the patient's own mind. They showed that the prodromal period is often measured in weeks rather than days, and gave the mean incubation period computed to the onset of earliest symptoms as ten weeks and to severe symptoms as 13 weeks (the time at which the disease was diagnosed in the above case). Morales Otero (1930), in experimental inoculation of volunteers, found that symptoms ordinarily causing attention took 28 to 35 days to develop, but he did not use the intravenous route. Unfortunately no clinical details are given in Meno Brito's paper, for it might be expected that symptoms develop more rapidly when infection occurs through the blood stream.

No evidence was found to incriminate milk as a source of infection in the above case, but there was, unfortunately, some difficulty and delay in tracing the two cows responsible for the patient's milk supply at the time. It would, however, seem to be pressing coincidence rather far to postulate a milk-borne source of infection developing

concurrently with exposure to infection through the blood of an individual known to have been in the bacteriaemic stage of the disease.

Summary

A case of brucellosis diagnosed 13 weeks after transfusion with blood from a donor in the bacteriaemic stage of the disease is described. If accepted, it is the first time that such an infection has been reported in this country, but such cases have been confirmed in Mexico.

Its occurrence illustrates the importance of questioning prospective donors about their health and rejecting those who might be in the incubation stage of an infectious disease.

The insidious onset of symptoms in undulant fever and the delay which may occur in the development of the antibody response are demonstrated.

I would like to express my thanks to Sir Weldon Dalrymple Champneys and to Professor Robert Cruickshank for their interest in this case; to Mr. Geoffrey Harvey, M.R.C.V.S., for investigating the patient's milk supply; to Dr. G. H. Tovey, of the Regional Transfusion Centre; and to the donor's medical attendant, Dr. R. C. Clifford, of Portishead, for his helpful notes. My thanks are also due to Dr. C. T. Andrews, physician to the West Cornwall Clinical Area, for permission to publish this case.

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Medical Memoranda

Lactation from Axillary Tail of Breast

Studying the literature on polymastia, which is taken to include supernumerary breasts, polythelia, and accessory aberrant or ectopic breast tissue in any form or situation outside the normal gland, one is impressed by the universal complaint of those few authors who have interested themselves in these interesting developmental anomalies that the subject has been somewhat neglected by medical writers.

In view of the relative rarity and the paucity of recorded cases, it was thought worth while publishing the following case, especially as no record of an identical one could be found, that of Anderson (1930) being closest in similarity.

CASE REPORT

A 25-year-old primigravida was on October 1, 1953, delivered normally at term of a live female infant weighing 7 lb. 2 oz. (3.2 kg.). On October 4 both breasts were engorged and tender. With expression, they were improved by the next day, but the patient became aware of moisture running down from the right armpit. On closer inspection this proved to be milk, and she was found to have bilateral moderately tender axillary breasts continuous with the main organs. Gentle pressure over the right-sided one caused milk to exude from a skin pore. The swellings were in the situation and relation of the axillary tails of Spence. The one on the left was of the same consistency but slightly smaller, and from it no milk could be expressed through the skin. There was no vestige of nipple or areola related to either swelling or elsewhere in the "milk line." The main breasts were normal, and satisfactory feeding was soon established, with subsidence of engorgement of the axillary tails. These had at no time caused gross discomfort.

Upon discharge from hospital on October 10, the axillary swellings were about half the size initially observed, and non-tender. Milk could still be expressed from the one on the right, but no longer ran out spontaneously. The infant had regained its birth weight. The patient was seen again on October 21, when the findings were the same as on the day of discharge.

Her mother, who has five children, had not been able to feed any of them, lactation failing in each case at the end of the lying-in period. She has no form of polymastia.

The patient has one sister, aged 24. She attended the antenatal clinic on October 27, when she was about 10 weeks pregnant, expecting her first child, and was found to have a unilateral swelling in the left axilla in the same situation as that of her sister. It was of the same consistency as the main breasts, but with no palpable communication with the one on that side, which was decidedly smaller than the right breast. On the right she has two rudimentary nipples in the "milk line," 3 in. (7.5 cm.) above and below the nipple of the main breast respectively.

COMMENT

Cases of axillary breast previously reported in which milk secretion occurred through a skin pore differ from the above case in that no connexion with the main breast was described (Anderson, 1930; Storey, 1936; de Cholnoky, 1939, 1951). More commonly, cases have been reported in which no milk-secreting duct opened on the surface of axillary breast tissue (Anderson, 1930; Christie, 1938; de Cholnoky, 1939, 1951; Masor, 1952). Some of these had been shown to communicate with the main breast by an isthmus of mammary tissue, others not. Most of these swellings had presented for the first time in the puerperium with the establishment of lactation, and most were very uncomfortable to the patient. De Cholnoky noticed that subjective complaint is much less where there is a connexion with the breast, as the secretion is discharged to and through the normal channels. None of the cases described, however, in which a communication with the main breast could be demonstrated, had milk coming through a skin pore over the axillary swelling.

In order that the presence of axillary breast tissue should not be missed, perhaps a suggestion that the best time to look for it is at or before the first post-natal examination would not be out of place. Storey's criteria are most useful.

I am indebted to Mr. C. W. F. Burnett, under whose care this patient was, for permission to publish the case report.

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Case of Kaposi's Varicelliform Eruption (Accidental Vaccinia)

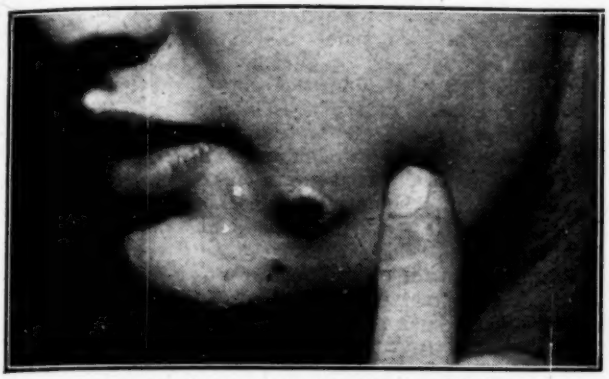
The following case shows the possibility of accidental vaccinia in a mother from contact with her vaccinated baby.

CASE REPORT

A 10-months-old child was vaccinated on April 2, 1953, in the upper and outer part of the thigh. This was successful, and the usual reaction followed. On April 18 the mother, who had never been vaccinated, developed a papule on the lower part of the left cheek and a sore in the left nostril in the naso-facial angle. A papule appeared on the following day on the posterior aspect of the terminal phalanx of the left index finger and also one on the interscapular part of the back. On April 22 the papules became pustular and there was a tendency towards umbilication. Considerable left infraorbital oedema developed with submaxillary adenitis. The temperature rose to 101.4° F. (38.6° C.) and the patient felt ill generally. As there had been several cases of smallpox in the North, a suspicion

arose about this case, and the medical officer of health was informed about it on April 23. A complement-fixation test of the pustular content was negative for vaccinia.

Dr. J. P. Marsden, Home Office Consultant, who saw the patient on April 24, was of the opinion that this was a case of accidental vaccinia, which he thought was rather rare. The accompanying photograph, taken on the same day, shows the typical appearance of the lesions and the facial oedema. A repeat complement-fixation test on egg membrane on April 27 proved positive for vaccinia, and so was



Photograph showing typical appearance of lesions.

an egg culture. Crusts began to form on the pustules on the 27th and the oedema was beginning to subside. The crusts separated about a week later, and small slightly depressed scars were left on the cheek and nostril.

This was a definite case of Kaposi's varicelliform eruption, as it fulfils the two requirements: (1) it was acquired by contact with a vaccinated person, and (2) it was definitely due to vaccinia virus, as proved by the complement-fixation test and egg culture.

COMMENT

Considering the large number of vaccinations performed, the number of reported cases of accidental vaccinia is comparatively very small; and although this case ran a benign course, the possibility of serious complications and sometimes of a fatal issue should be borne in mind. Finn *et al.* (1952) report a case of accidental generalized vaccinia in a child 6 months old, contracted from another child admitted thirteen hours previously into a cot adjacent to the patient's and who had had an active vaccination sore for ten days. The patient developed lesions twelve days later and died after ten days, in spite of treatment. Good and MacLachlan (1950) report a case of accidental generalized vaccinia in a boy of 6, who played in bed with a recently vaccinated infant; the boy died in hospital nine days later from vaccinia.

Vaccinia may also develop in a person who has been vaccinated, as illustrated in a case reported by Blattner (1951) of a child who developed impetiginous lesions after a smallpox vaccination. Vaccinia changes were demonstrated in the material taken from the lesions fifteen days after vaccination. According to Blattner *et al.* (1946), humoral antibodies to vaccinia begin to appear at the end of the first week following cutaneous vaccination; they increase in amount during the second and third weeks and reach their maximum at the end of the third week.

Bigler and Slotkowski (1951) maintain that the microscopical changes in vaccinia are identical with those of variola. It is believed that vaccinia and variola are caused by the same virus. When variola is inoculated into cattle and carried through several transfers in these animals the virus (variola) becomes attenuated and approaches the nature of vaccinia. The changes in the Malpighian layer of the epidermis consist of swelling, cytoplasmic degeneration and liquefaction, formation of multilocular vesicles, and eosinophilic inclusion bodies—Guarnieri's bodies—which are swollen nucleoli that have migrated from the nucleus into the cytoplasm.

It would therefore seem advisable to issue warnings about the possibility of accidental vaccinia from undue contact with recently vaccinated persons. The risk appears to be greater in people who have never been vaccinated.

I would like to express my thanks to Dr. J. P. Marsden, consultant, Joyce Green Hospital, for his kind help and advice about the case, and to Dr. J. C. Birchall, assistant medical officer of health for Croydon, for his great interest in the case and for the repeated complement-fixation tests and cultures done through him in the virus laboratories.

A. FRY, M.R.C.S., L.R.C.P., D.M.R.

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Torsion of Gall-bladder

Torsion of the gall-bladder is a relatively rare emergency, which can occur only in those cases in which the gall-bladder is suspended from the liver by a mesentery. Bean and Symond (1953), in a review of the literature, found 100 cases reported up to 1953. The first case was reported by Wendel in 1898. In his case there was a floating gall-bladder, with a long cystic duct, and a floating kidney, with cholelithiasis and perforation of the gall-bladder. Brewer (1899), in 100 consecutive dissections, found only five cases in which the gall-bladder had a mesentery. A mesentery is only one of the several possible congenital anomalies of the gall-bladder. Recht (1952) reported a case in which he found a double gall-bladder, one part of which had undergone torsion. According to Maingot (1948) most cases of torsion occur during later life—that is, at the age of 60–70. Short and Paul (1934) estimated that 84% of cases occur in females. A further case is here presented.

CASE REPORT

On admission the patient, a woman aged 71, complained of severe abdominal pain, mostly in the upper part and down the right side of the abdomen. She stated that the pain had started in her neck, and seemed to pass down her chest to settle in her stomach. She had vomited several times prior to admission. There were no other symptoms of note. Her own doctor had diagnosed angina pectoris. She stated that for the past two years she had had severe breathlessness on slight exertion.

On examination she was noted to be a frail, thin, ill-looking old lady, obviously in severe pain. She was edentulous. The tongue was clean and moist. No abnormal physical signs were detected on examination of her chest, apart from the fact that the apex beat was 1¼ in. (3.8 cm.) outside the nipple line. There was no abdominal distension; only slight movement with respiration was noted, and the abdomen was rigid down the right side, where it was also very tender. A distinct zone of maximal tenderness was noted about 1½ in. (3.8 cm.) above and to the right of the umbilicus, this area being approximately 1 in. (2.5 cm.) in diameter. No mass could be felt and no loss of liver dullness was detected. Her temperature was 99° F. (37.2° C.), pulse 95 and regular, and blood pressure 290/120.

A provisional diagnosis of perforated duodenal ulcer was made and the patient prepared for operation.

A laparotomy was performed through a right upper paramedian incision. When the peritoneum was opened a pyriform black mass presented, which, on further inspection, was found to be a gangrenous gall-bladder. The gall-bladder was suspended from the liver by a mesentery and had undergone torsion in an anticlockwise direction. The mesentery extended from near the fundus to the proximal part of the cystic duct and was ½ in. (1.3 cm.) across its widest part. The torsion involved the proximal half of the cystic duct, which was longer than usual. The gall-bladder was easily removed after the cystic duct and artery had been identified

and divided, only the mesentery remaining to be dealt with. A drainage tube to the gall-bladder bed was brought out through the upper part of the wound, which was then closed in layers. The patient's general condition remained satisfactory during and after the operation. Her general condition improved considerably during the next twenty-four hours; she continued to make good progress until her discharge from hospital on the fourteenth post-operative day.

COMMENT

One very interesting clinical sign in this case was the distinct zone of acute tenderness over a small area. This had previously been noted by Case (1951). The gall-bladder was relatively free and mobile, but when torsion occurred it became relatively fixed and firmly abutted against the anterior parietal peritoneum. The small zone of maximal tenderness therefore corresponds fairly accurately to the area in contact with the gangrenous gall-bladder.

The patient's convalescence was without incident, which is not only gratifying but interesting; Arthur (1937) estimated the mortality to be 16%.

I wish to thank Mr. G. Townsley for permission to report this case.

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Successful Femoral Embolectomy 17 Days After Acute Coronary Occlusion

Instances of successful removal of large emboli are not numerous. In a series of 19 femoral or iliac embolectomies performed during a five-year period (1946–51) at the Cook County Hospital (Klingensmith and Theis, 1952) only one patient was discharged with a surviving embolectomized leg, 15 died in the hospital, and 3 were discharged with an above-knee amputation. There are other reports in the literature claiming better results than this, but, even so, the percentage of successes is small.

A common cause of peripheral embolism is mural thrombus following myocardial infarction. Boas and Boas (1949) state that such thrombi are found in about 50% of cases of myocardial infarction that come to necropsy. Hellerstein and Martin (1947) found that the incidence of clinically detectable thrombo-embolic lesions in myocardial infarction in 1,600 cases was 11.5%. The incidence of embolism in the extremities was 5.5%.

CASE REPORT

A woman aged 64 was admitted to hospital on March 24, 1953. She was brought in by ambulance after an attack of severe substernal pain, which came on suddenly while she was walking.

On admission she was not shocked; pulse 82, irregular owing to extrasystoles; blood pressure 145/85. The heart sounds were normal, no murmurs or pericardial rub were heard, and there were no signs of cardiac failure. A diagnosis of myocardial infarction was made. This was confirmed by an electrocardiogram, which showed a recent anterior coronary occlusion. The patient was treated by rest in bed. No anticoagulants were used.

Satisfactory progress was made, apart from some chest infection, until April 10, when she complained of sudden onset of pain, first on the left side of the abdomen, and then, shortly after, by very severe pain in the right leg. On examination the right leg was pale, cooler than the left, and no arterial pulses were palpable. After ten minutes the limb had become mottled and blue. She was treated with morphine, ¼ gr. (16 mg.), tolazoline hydrochloride, 50 mg. intravenously; heparin, 10,000 units; and ethyl biscoumacetate, 900 mg. After one hour the limb was still cold and cyanosed, but the pulsation of the external iliac artery had become palpable.

Three hours after the onset of pain an operation was carried out under local analgesia. No premedication was given, as the patient had had morphine for her pain.

One per cent. procaine was infiltrated and an incision made over and in line with the right femoral artery, in the femoral triangle. The artery was freed. Its wall was not thickened or calcified. Tapes were applied around the artery, which was opened between the tapes. Several small clots were removed. By releasing the pressure on the proximal tape, a thrombus 50 mm. long shot out of the lumen of the vessel. A ureteric catheter was inserted in the distal part of the artery, and 5 ml. of sodium citrate and 1 ml. of heparin was injected through the catheter. The incision was closed with an arterial suture. The sheath of the artery was not closed. The deep subcutaneous tissues were closed with plain catgut. The skin wound was closed and a firm bandage applied.

At the end of the operation there was a marked improvement in the circulation. The pulsation of the popliteal, dorsalis pedis, and posterior tibial arteries was palpable. The limb, however, remained mottled and cyanosed for some 12 hours post-operatively. At the end of the operation the patient complained of pins-and-needles in the toes.

An intermittent venous occluder was applied to the limb in the mid-thigh and left on for 24 hours, after which it was discontinued, as the circulation appeared to be normal.

Heparin therapy, 10,000 units eight-hourly, was given for 24 hours, and ethyl biscoumacetate, controlled by prothrombin times, was given daily for three and a half weeks.

Post-operative progress was uneventful. The patient was allowed up after four weeks, and was discharged quite well on May 16, five weeks after the embolectomy.

Our thanks are due to Mr. J. Thompson Fathi for his help and encouragement in the preparation of this article, and to Dr. P. J. Mills for his permission to publish this case.

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J. K. N. YEARSLEY, M.B., B.S.

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The Diagnosis of Mongolism in Infancy: Small Auditory Meatus

It was pointed out in a previous publication (Wallis, 1951) that the diagnosis of mongolism in infancy is made easier by the observation of Brushfield's spots. In Brushfield's (1924) own words these are: "Speckled white or very light yellow clearly defined pin-points near the outer margin, generally placed at regular intervals in a ring and appearing to be placed on the iris. I have not been able to find these in the brown iris."

Observation of a further 10 cases has confirmed my surmise that at birth all mongols have blue eyes and show Brushfield's spots. Unfortunately some normal children show the spots as well. However, at least if a newborn infant has not got them we may be sure that it is not a mongol.

Another sign which has come to light is the smallness of the external auditory meatus. In all 10 cases it has been found impossible to insert an auriscope speculum of 3 mm. diameter into the ear, and in fact in not one of them could the drumhead be seen on either side. This is merely an expression of the general lack of development of the basal bones of the skull, but it has not apparently been recorded previously.

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Reviews

THE BRAIN IN EPILEPSY

Epilepsy and the Functional Anatomy of the Human Brain. By Wilder Penfield, O.M., C.M.G., M.D., F.R.S., and Herbert Jasper, M.D., C.M. Chapter XIV by Francis McNaughton, M.D., C.M. (Pp. 896+xv; 314 illustrations, 8 coloured plates. £5 15s.) London: J. and A. Churchill Ltd. 1954.

This large volume, which is a sequel to *Epilepsy and Cerebral Localization,* written by Penfield and Erickson in 1941, presents material collected at the Montreal Neurological Institute since Dr. Jasper joined Dr. Penfield there in 1938. It deals, through detailed fact and copious illustrations, with the experimental neurosurgery and human electrophysiology that these workers have undertaken in that time. The historical background of their subject is also summarized.

The importance of the work can be judged from the fact that before the second world war, when this work began, to record directly from the cortex of man was a remarkable event and to stimulate it electrically an isolated experiment. Dr. Penfield's greatest achievement has probably been to secure such close co-operation from hundreds of patients who, having simply local analgesia of the operation area, have been able to describe their experiences under cortical stimulation and to help in hours of clinical and electrophysiological recording. Everyone who is concerned with cerebral physiology will wish to study the detailed observations included in this volume. As the methods and circumstances of recording are also fully described they can be collated and interpreted by the reader himself. This must have been the authors' intention, as they suggest in the preface, for such a wealth of fact, including the invocation by stimulation of every sensation, most movements, and some of the emotions which man experiences, cannot be interpreted by one school or in one era.

Roughly, the first part of the book considers epilepsy, especially from the neurosurgeon's point of view; the last sections deal with clinical, surgical, and electrophysiological techniques; and the larger middle portion presents the observations made by Dr. Penfield and his team of workers. The method employed is to illustrate a chapter with relevant case histories, though these differ from those usually found and from clinical texts in their intimate nature. They include photographs of the stimulated brain at operation, with verbatim accounts of important observations made by the patient or one of the team. Epileptic experiences are reproduced and past events invoked by cortical stimulation, and the psychologist and philosopher can make of it what he will. That this wealth of accumulated observations cannot yet be logically integrated is apparent in those sections of the book dealing with memory or the concept of a centrencephalic mechanism.

Although "the dedication of this book to Hughlings Jackson is prompted by the knowledge that many of the conclusions reported here were long ago the surmise of the founder of the English School of Neurology," there is surely still a long way to go before the facts presented can lead to consistent conclusions which would have been acceptable to him. It is this rather than any conclusions suggested which makes the book so valuable and provocative. It is particularly welcome to those who have followed Dr. Penfield's work through the years, for its publication coincides with his retirement from the Chair at McGill.

DENIS WILLIAMS.

ATOMIC MEDICINE

Atomic Medicine. Edited by Charles F. Behrens, M.D. Second edition. (Pp. 632+xiv; illustrated. 84s.) London: Baillière, Tindall and Cox, Ltd. 1953.

The development of atomic energy has introduced entirely new medical problems in connexion with many of its potential uses and abuses. The atomic industries require health precautions of a novel kind which are already highly developed; the clinical and experimental use of isotopes and of nuclear radiations is constantly yielding new methods of investigation and therapy; and nuclear weapons can cause widespread radiation hazards in a variety of different ways. This is the broad subject, or rather the group of subjects, which is designated "Atomic Medicine" and discussed in detail in the second edition of a valuable work originally produced in 1949, largely by members of the medical and scientific staff of the United States Navy.

In many ways this is an important book, and particularly for its account of severe radiation injury and for the practical survey of health precautions in atomic industries. These are unfamiliar fields of medical responsibility on which it is useful to have the available information so well assembled. The section on particular isotopes is sometimes rather more superficial, perhaps because a large field, which has been the subject of several excellent textbooks, is here attempted in a few chapters. The bibliography of some chapters shows signs of inadequate revision since the 1949 edition, although in the work as a whole both the references and the tabulated material are of considerable value for reference. Inevitably in a composite work by different authors there are reduplications and omissions, but the planning and editing are usually careful and good, considering the rather disconnected subjects that the book attempts to cover. Parts of the chapter on "Survival Methods in Atomic Disaster" are somewhat trivial or naive, but a picture emerges which is coherent and practicable. Readers of this book will, incidentally, enjoy the conception of "the World War II figure of the 'air-raid warden' idly swinging his baton or flash-light during an 'alert' or a 'blackout,'" and agree that this figure needs replacing.

This book is important to those concerned with nuclear radiation and shows clearly to what extent the biological hazards are serious and are controllable.

E. E. POCHIN.

TROPICAL MEDICINE

A Manual of Tropical Medicine. Edited by Thomas T. Mackie, M.D., Colonel M.C., A.U.S.(Retired), George W. Hunter III, Ph.D., Colonel M.S.C., U.S.A., and C. Brooke Worth, M.D. Second edition. (Pp. 907; illustrated. 60s.) Philadelphia and London: W. B. Saunders Company. 1954.

In tropical medicine the size of a textbook has always been of special importance, for those working in this field are likely to travel and to need a textbook while doing so. A volume which covers the subject-matter in the minimum of space has therefore a very special appeal. This one is intended not to be a voluminous work of reference, but to contain sufficient detail to be an authoritative guide to those practising tropical medicine. It is of readily transportable size, it is packed with up-to-date, well-presented information of a high degree of accuracy, and it covers all the diseases likely to be encountered by a practitioner in the Tropics. To deal in detail with such a wide range of diseases is now beyond the scope of any small group of authors, and the editors are to be complimented on calling upon twenty-four contributors to help them.

As is right in an American publication, special emphasis is given to diseases of the tropical parts of the New World. American enthusiasm for the use of antibiotics has in places been allowed to obscure the value of older but highly effective standard remedies. Thus it is said of emetine bismuth iodide, which outside America is regarded by the majority of workers as being the most effective drug for the treatment of amoebic dysentery, that it and similar preparations are now seldom necessary or desirable.

Diseases of military importance are also given special attention, and the fact that the work is up-to-date is emphasized by a section on the recently described epidemic haemorrhagic fevers. Unfortunately nutritional diseases, which to tropical peoples are of tremendous importance, are dealt with inadequately. Sprue is classed as a nutritional disease, and to it and all other diseases in this section a mere

twenty-two pages are devoted. To macrocytic anaemia and kwashiorkor, which together probably affect many millions of tropical inhabitants, less than one page is devoted, as compared with six pages dealing with epidemic haemorrhagic fever, of which only a few hundred cases have so far been reported. In spite of these shortcomings, however, the book is a good one, and it is certain to be warmly received. The printing, paper, and the 304 illustrations are excellent.

A. W. WOODRUFF.

AN ANATOMICAL ATLAS

Atlas of Descriptive Human Anatomy. By J. Sobotta. Edited and translated by Eduard Uhlenhuth, Ph.D. Volume III. Blood vessels—Nervous system—Sense organs—Integument and Lymphatics. 5th English edition. (Pp. 390+xix; 164 coloured and 142 black and white illustrations on full page plates and additional 67 partly coloured figures in the text. £6 5s.) New York: Hafner Publishing Company Inc. London: Lange, Maxwell and Springer. 1954.

The late Professor Sobotta was not only a distinguished teacher of topographical anatomy but also famous for his many accurate investigations in the field of early mammalian development. His contributions to the study of the maturation, fertilization, and cleavage of the ovum of the mouse are regarded as classics. The present volume of his *Atlas of Anatomy* is the fifth English edition of this now famous book, first produced in German in 1904. After an absence of nearly ten years this edition will be welcomed by teachers and students alike. The first English edition was prepared by Professor McMurrich, of the University of Toronto; the fifth has been edited and translated by Professor Edward Uhlenhuth, of Baltimore. As in earlier editions, arteries, veins, and nerves are shown together in a single illustration. This has many advantages in that the student is able to see the relationships of these structures as they are found in the body and not as separate entities when they are illustrated in separate diagrams. The only criticism I would make is that in several of the illustrations too many fine branches of nerves and arteries have been shown, and in certain figures such detail obscures the essentials. The text is clear and concise. The atlas is one of the best available to-day and can be recommended with confidence.

W. J. HAMILTON.

CARDIOVASCULAR SURGERY

Cardiovascular Surgery. By Gerald H. Pratt, M.D., F.A.C.S. (Pp. 843; 358 illustrations, 4 coloured plates. £5 10s.) London: Henry Kimpton. 1954.

The rapidly increasing scope of cardiovascular surgery makes it difficult to condense the sum total of our present knowledge into a reasonable space. Dr. Pratt has, however, achieved this end and has given a concise and yet comprehensive account of the subject in one volume. Diseases of the heart and blood vessels are becoming increasingly important in a society whose expectation of life is steadily improving, and surgery in this field has, therefore, a similarly increasing significance, even though only a proportion of the disorders are amenable to treatment.

The book is divided into sections, starting with a classification and the general examination of the patient, and passing via anaesthesia, fluid balance, and the like to surgery of the heart, of the arteries, of the veins, and of the lymphatics and thence to special technical procedures. Each section has an adequate, though not complete, bibliography, and there are numerous well-chosen illustrations to amplify the text.

In the chapters on heart surgery the description of mitral stenosis and its treatment is very satisfactorily handled. Other cardiac lesions suitable for surgery are more briefly, though adequately, discussed. Occlusive arterial disease is described in detail, and not only are surgical procedures well illustrated, but conservative measures, including dietary instructions, are given in a practical manner. The treatment of threatened and actual diabetic gangrene is similarly dealt with. Arterial thrombosis, embolism, aneurysm, and arteriovenous fistula are discussed with a wealth of detail that is based on the author's considerable experience and makes valuable reading.

There is little that the author has omitted in his comprehensive survey of the whole subject, and if there is a certain unevenness in some sections it is a very minor criticism of a book which must well remain for some time a standard work on this complex subject.

T. HOLMES SELLORS.

ANGIOGRAPHIC STUDY OF PATHOLOGY

Atlas Postmortaler Angiogramme. By J. Schoenmackers and H. Vieten. (Pp. 203; illustrated. D.M. 57.) Stuttgart: Georg Thieme. 1954.

This most original volume consists of a series of radiographs taken from cadavers after injection of the blood vessels with opaque material, thus enabling a large number of pathological conditions to be studied from a very unusual point of view, often with surprising results. The technique employed is described in detail, and the superb radiographs which have been obtained are a proof of its success. The skill and labour involved must have been very great, but they were fully justified by the result.

Every region of the body has been covered and in a large variety of pathological conditions. Each print is accompanied by a full description and frequently by an explanatory diagram. At the end of each section a summary is given in four languages, but in general the plates speak for themselves. They number in all 131, and in every case the production is perfect. Perhaps the most interesting are those of the lungs and of the heart. Angiograms of the lungs include acute infection, asthma, emphysema, tuberculosis, silicosis, and carcinoma, while those of the heart are devoted to the coronary arteries, showing variations of the utmost significance.

The importance of this laborious study as a background will be realized when the great development of such radiographic study of the living in clinical practice is considered. Comparison of a clinical angiogram with the corresponding plate in this atlas should greatly facilitate an appreciation of its meaning and assist in diagnosis. But perhaps it is the pathologist himself who will best appreciate these superb plates and the perfect technique which their production demanded.

HENRY SOUTTAR.

PHYSIOLOGY OF THE MOUTH

The Physiology of the Mouth. By G. Neil Jenkins, M.Sc. (Liverpool), Ph.D. (Cantab.). (Pp. 288; 53 figures. 30s.) Oxford: Blackwell Scientific Publications. 1954.

The physiology of the mouth and dental tissues has not in the past received the attention it deserves, and it is most useful to have a modern work on the subject with very excellent references collected together in one volume. Although written primarily for undergraduate students, for whom it will be most valuable, its greatest value will probably be to the postgraduate student and those interested in research in this field, since it deals with current aspects of research which may be somewhat beyond the basic training of the dental student. The author is to be congratulated on producing a most interesting book.

A. MACGREGOR.

Alumni Cantabrigienses, compiled by J. A. Venn, is a biographical list of all known students, graduates, and holders of office at the University of Cambridge from the earliest times to 1900. The four volumes of Part I covered from the earliest times to 1751, and the sixth and final volume of Part II (from 1752 to 1900) has recently been published, the names in this volume running in alphabetical order from Square to Zupitza. The compiler and publishers (Cambridge University Press) are to be congratulated on successfully completing this immense biographical task. The cost of the final volume is the same as that of its predecessors, £10 10s.; and the whole set of ten volumes can be bought for £89 5s.

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ADRENALECTOMY FOR BREAST CANCER

Most of our present-day knowledge of the aetiology of breast cancer is based on animal experimentation. Although as early as 1919 Loeb and his co-workers had demonstrated the carcinogenic potency of the ovaries in mice with a high susceptibility to breast cancer, it was not until 1932 that Lacassagne[1] finally established the relationship between the ovarian factor and mammary neoplasia. His discovery that oestrone induces breast cancer in male mice, which normally never develop it, was important because oestrogen was proved to be the first substance originating in the body which causes cancer. Further research into the biological activities of the sex hormones showed that administration of the male sex hormone could prevent the development of mammary cancer in female mice belonging to high cancer strains. These results produced a wave of optimism, for many then took the view that cancer which arose in organs under endocrine control could be prevented, or at least held in check, by administering a hormone antagonist.

Loeser[2] was one of the first in the clinical field to try testosterone therapy in cases of inoperable breast cancer, and Adair and Hermann,[3] among others, reported later on this form of treatment. Haddow and his colleagues,[4] Nathanson,[5] and Stoll and Ellis,[6] relying on the depression of pituitary function and hence inhibition of ovarian secretion in preference to a hormone antagonist, used massive doses of oestrogen for treatment of breast cancer in women. These different forms of treatment gave variable results. Some breast neoplasms were temporarily held in check, others failed to respond. Apparent recovery, however, was extremely rare, and reactivation of tumour growth invariably occurred. This is probably due to the fact that when an endocrine imbalance is created, either by the surgical removal of an organ or by the administration of a hormone, the anterior pituitary gland stimulates other endocrine organs to make up for the deficiency. Thus the adrenal cortex might be stimulated to secrete both oestrogens and androgens, the ovary to secrete androgens, and the sertoli cells of the testes to secrete oestrogens sufficient in amount according to some authors[7] to induce cancer of the male breast.

Huggins,[8] working on the assumption that steroid hormones of the adrenal cortex might be responsible for reactivation of tumour growth, was the first to advocate bilateral adrenalectomy combined with castration in cases of breast cancer which had failed to respond to other forms of hormone treatment. He has recently published a review[9] of these results, and Sir Stanford Cade makes another important contribution in the opening pages of this *Journal* to the knowledge being gained about this method of treatment. Nearly 20% of deaths from cancer in women in Great Britain are from mammary carcinoma, and the importance of the recent work must be judged against this background, but both Huggins and Cade are emphatic in claiming only that this form of therapy controls the growth of the disease rather than cures it. The operative mortality, considering the physical state of the patients, is very low. Post-operative adrenal insufficiency is made good by the administration of cortisone acetate. Those with skeletal metastases obtain immediate relief from pain, and begin to gain weight. Cade reports on 56 patients who had bilateral adrenalectomy for disseminated breast cancer. The results have been good in 60% of these cases, including 23% which showed remarkable improvement, regression of palpable lesions being accompanied by reossification in skeletal metastases. Survival so far has been up to 21 months. Huggins, in his recent review, reports that out of 17 patients subjected to adrenalectomy for mammary cancer in May, 1951, there was only one post-operative death. Six patients were alive two to three years after the operation, and secondary deposits had regressed in all of them. One patient, three years after the operation, has apparently recovered and has returned to work. Unfortunately only some breast cancers respond to this treatment: in fact there appear to be three distinct types, none of which with any certainty can be histologically diagnosed. There are those tumours which are adrenal-dependent for sustained growth and respond to adrenalectomy. The second type are those which are ovary-dependent, and these likewise respond to the combined operation of castration and adrenalectomy. Growths of the third type, which are rarer, are hormone-independent and fail to respond to any form of hormone therapy. This resistance may possibly be due to a factor of hypophyseal origin. Since there are no reliable means

[1] *C.R. Acad. Sci. Paris*, 1932, **195**, 630.
[2] *British Medical Journal*, 1940, **1**, 479.
[3] *Ann. Surg.*, 1946, **123**, 1023.
[4] *British Medical Journal*, 1944, **2**, 393.
[5] *Endocrine Aspects of Human Cancer*, 1947, Academic Press, p. 261.
[6] *British Medical Journal*, 1953, **2**, 796.
[7] Burrows, H., and Horning, E. S., *Oestrogens and Neoplasia*, 1952, Oxford, p. 90.
[8] *J. Urol.*, 1952, **68**, 875.
[9] *J. nat. Cancer Inst.*, 1954, **15**, 1.

whereby it can be ascertained beforehand whether a breast cancer is hormone-dependent, the results obtained by adrenalectomy must remain unpredictable.

Admittedly there are many gaps in our present knowledge of the functional inter-relationships of the endocrine system, and until a method of controlling the activity of the anterior pituitary gland can be found the treatment of mammary cancer by hormone therapy will remain incomplete and often unsatisfactory. Nevertheless, as Sir Stanford Cade rightly states, "No method of treatment has as yet achieved control of disseminated, widespread, and often terminal stages of breast cancer comparable to that following adrenalectomy."

YELLOW FEVER IN AFRICA

Until the early years of this century yellow fever had been one of the great plagues of the Tropics and sub-Tropics for more than two hundred years. In 1900 the discovery of the transmission of the disease by the mosquito *Aëdes aegypti* was the first of many important advances, and since then epidemiological investigations have led to methods of control so efficient that a major epidemic need no longer occur anywhere. At first it was believed that yellow fever was essentially an urban disease carried only by *A. aegypti*, but experiments in 1928 showed that certain other mosquitoes could transmit it under laboratory conditions. In 1932, in Brazil, a rural epidemic was studied in which *A. aegypti* was not the vector, and similar outbreaks, in which secondary cases were much less common than in urban areas, were noted in populations living in or close to uncut forest. These facts suggested the existence in the jungle of a transmission cycle involving a mosquito and an animal host, human infection being only incidental. This theory was supported by the presence of protective antibodies in locally captured monkeys, and in 1944 it was confirmed by the recovery of yellow fever virus from a wild marmoset and from the forest mosquito *Haemagogus spegazzinii*.[1]

In Africa the study of rural yellow fever has been actively pursued in the Bwamba forest of Uganda since 1936, and there a similar epidemiological pattern was found. In Brazil jungle yellow fever was contracted mainly in virgin forest, but in the Bwamba area infection mainly occurred in farm clearings in the forest. As the local epidemiology was elucidated it was found that the main sylvan hosts were monkeys living in the treetops and never descending to ground level. The virus was carried between them by *Aëdes africanus*, a night-biting, treehole-breeding species which also lived in the tree canopy.[2] A further link was required to bring the disease to man. This was provided by other species of monkeys which rested in the trees at night where they could be infected by *A. africanus*, but during the daytime they raided farms and were then bitten by *Aëdes simpsoni*.[3] This species fed during daylight on both monkeys and man, and it bred in the leaf axils of banana and colocasia plants in the edges of clearings and forest fringes. By this chain of transmission in the Bwamba forest the virus is transported from the tree canopy to man at ground level and can then be carried to towns, where man-to-man transmission by *A. aegypti* may result. The epidemiology requires clarification, however, in some other African areas where there are insufficient monkeys to support continued transmission of the virus and where a prolonged dry season eliminates or reduces the vector population to a very low level for some months. In this connexion it may be significant that protective antibodies have been demonstrated in wild African lemurs living in savannah country. It has also been shown that certain mites and ticks can ingest and conserve yellow-fever virus for days or weeks, though transmission by their bites has not been effected.[4] Infection of a sandfly has also been recorded in nature.[2]

Modern methods of controlling yellow fever are based on the specific epidemiology of the disease. In urban areas transmission is prevented by eliminating *A. aegypti* by means of larval control and residual insecticidal spraying of houses, or by reducing its density below that necessary for epidemic spread. Control of the sylvan form of transmission is a much more difficult problem because no practical means exist of attacking the insect vectors or the monkey hosts. A method of prophylactic treatment of man by live attenuated virus is, however, available. Two vaccines are in use, 17D vaccine produced by the Rockefeller Foundation by culture in denervated chick embryo, and a vaccine prepared by the Institut Pasteur in Dakar from infected mouse brain. The former is administered subcutaneously, while the latter can be given either subcutaneously or by scarification, in which case it may be combined with smallpox vaccine. The period of protection given by both preparations is officially recognized as six years, and is probably longer. Reactions following 17D vaccine are rare and mild and many millions of people have been protected by it. The Dakar vaccine in a small proportion of cases causes neurotropic reactions, but it has been used extensively in French West Africa,

[1] Laemmert, H. W., and Ferreira, L. de C., *Amer. J. trop. Med.*, 1945, **25**, 231.
[2] Smithburn, K. C., *et al.*, *Ann. trop. Med. Parasit.*, 1949, **43**, 74.
[3] Mahaffy, A. F., *et al.*, *Trans. roy. Soc. trop. Med. Hyg.*, 1942, **36**, 9.
[4] Davis, N. C., *Amer. J. trop. Med.*, 1933, **13**, 547.
[5] Breteau, H., *Bull. Wld Hlth Org.*, 1954, **11**, 453.

where more than 38 million doses have been administered.[2] The results of this method of control have been excellent. In treated areas sylvan yellow fever is no longer seen in the human population, or occurs only exceptionally, usually in remote localities in persons who have escaped vaccination. The spread of sylvan yellow fever from the permanent foci to urban areas can thus be prevented, while *Aëdes* control in towns obviates epidemic transmission if the disease is introduced and reduces the risk of its export to other parts. *Aëdes* control on airfields and the use of insecticides in aircraft are necessary to prevent the carrying away of infected vectors. There still remains a risk of spread from centres so far unrecognized, but this is becoming more remote as immunity surveys delineate more accurately the endemic zones and foci of the disease.

SEQUELAE OF ERYTHROBLASTOSIS

Rhesus incompatibility is a risk run in about one-eighth of pregnancies, with harmful antibodies developing only in about one in two hundred. A further 20% of maternal sera contain anti-A or anti-B agglutinins incompatible with the infant's cells,[1] though haemolytic disease complicates only a small proportion of these cases. The most important complication of the disease is kernicterus; before the introduction of exchange transfusion it was reported in up to 30% of cases. Deep jaundice is accompanied by such signs as head retraction, eye rolling, squint, opisthotonos, hyperpyrexia, convulsions, and terminal respiratory failure. A hypotonic phase usually follows in the one-third who survive the acute stage, and this is generally succeeded by choreo-athetosis with or without deafness, in some cases accompanied by mental retardation. Minor abnormalities may also occur in jaundiced babies in the first few days, such as lethargy, reluctance to feed, and vomiting, sometimes with increased muscle tone or mild cerebral irritation, but such cases apparently carry a better prognosis, and several have been reported as being entirely normal at the age of a year.[2] A correlation between hyperbilirubinaemia and kernicterus was suggested by the identification of the staining pigment as bilirubin,[3] the discovery that the indirect type has a special affinity for the basal ganglia,[4] and by the experimental kernicterus produced by bilirubin infusion in animals.[5 6] Thus it has been established that kernicterus is rare if the serum bilirubin level remains below 18 mg. per 100 ml.[7] Exchange transfusion removes the source of approximately 1,000 mg. from the infant's blood and is therefore indicated if there is anaemia with cord haemoglobin below 15 g. per 100 ml., prematurity, or a history of a previous affected sibling; otherwise it is given if the bilirubin level rises above 18 mg. per 100 ml.[1] Armitage and Mollison last year reported[2] the results of a therapeutic trial encompassing about 450 liveborn infants wherein alternate anaemic babies with haemoglobin below 15.5 g. per 100 ml. were treated by exchange and simple transfusion. In the whole series there was still a 9% overall mortality from kernicterus up to the end of the first year, while a further 5% of 350 tested survivors were backward or showed neurological abnormality in a 1–3-year follow-up. In spite of effecting a reduction of 50% in the mortality from kernicterus, a single exchange transfusion did not avert this complication in 11% of anaemic full-term babies and 35% of anaemic premature babies. Better results are attained by repeating the exchange transfusion if further jaundice or hyperbilirubinaemia develops, which measure succeeded in reducing the immediate mortality from kernicterus to 2% in 250, mostly full-time, sensitized babies.[8] One case is also worth recording of an infant who received exchange transfusion subsequent to the onset of acute kernicterus and later apparently developed normally.[9]

Apart from the sequelae of frank kernicterus, there remains the question whether blood group incompatibility can lead to mental retardation in cases where there is no evidence of acute or subsequent neurological damage. Creak and Durward[10] found normal distribution of intelligence in the 45 physically normal survivors of 50 children originally treated at Great Ormond Street for the neonatal effects of rhesus incompatibility. Another method of investigation has been to determine the rhesus blood groups of undifferentiated mental defectives and their parents. Yannet and Lieberman,[11] comparing the blood groups of neurologically normal mental defectives whose defect was of unknown aetiology with the blood groups of their mothers, found an increased incidence of rhesus incompatibility as compared with aments of known aetiology, but numerous later investigators[12–14] have failed to confirm these findings or similar ones for ABO incompatibility.[15] More satisfactory have been two

[1] Mollison, P. L., *Recent Advances in Paediatrics*, edited by D. Gairdner, 1954, Chap. 5, p. 110. Churchill, London.
[2] Armitage, P., and Mollison, P. L., *J. Obstet. Gynaec. Brit. Emp.*, 1953, **60**, 605.
[3] Waters, W. J., *et al.*, *Pediatrics*, 1954, **13**, 319.
[4] Claireaux, A. E., *et al.*, *Lancet*, 1953, **2**, 1226.
[5] Küster, F., and Krings, M., ibid., 1950, **1**, 979.
[6] Day, R., *Amer. J. Dis. Child.*, 1947, **73**, 241.
[7] Mollison, P. L., and Cutbush, M., *Blood*, 1951, **6**, 777.
[8] Diamond, L. K., *et al.*, *Pediatrics*, 1952, **10**, 337.
[9] Abelson, N. M., *Med. Clin. N. Amer.*, 1953, **37**, 609.
[10] *Gt. Ormond Str. J.*, 1953, **6**, 81.
[11] *Amer. J. ment. Defic.*, 1944, **49**, 133.
[12] Zwerling, I., *et al.*, *Amer. J. Dis. Child.*, 1951, **82**, 7.
[13] Böök, J. A., *et al.*, *Amer. J. hum. Genet.*, 1949, **1**, 66.
[14] Gilmour, D., *J. ment. Sci.*, 1950, **96**, 359.
[15] Yannet, H., and Lieberman, R., *Amer. J. ment. Defic.*, 1945, **50**, 242.
[16] *J. Pediat.*, 1950, **36**, 342.
[17] *Pediatrics*, 1954, **13**, 333.
[18] Evans, P. R., and Polani, P. E., *Quart. J. Med.*, 1950, **19**, 129.
[19] Fisch, L., and Osborn, D. A., *Arch. Dis. Childh.*, 1954, **29**, 309.
[20] Gellis, S. S., *et al.*, *Amer. J. Dis. Child.*, 1954, **88**, 285.

well-controlled mental and neurological follow-up studies by Day and his colleagues on babies known to have had haemolytic disease of the newborn without having developed clinical signs of acute kernicterus or any subsequent neurological deficit. Gerver and Day[16] reported a normal average I.Q. of 102.7 in a series of 68 such babies, but the sibling controls were on the average 11.8 points higher, while five of the affected babies and none of the siblings had I.Q.s below 70. In a second study, after the introduction of exchange transfusion, Day and Haines[17] reported that deeply jaundiced babies with severe anaemia suffered a significant depression of 23 points, as opposed to the entirely normal intelligence found in babies with absent or moderate jaundice. This danger from deep jaundice, which was also present to a lesser degree in cases without severe anaemia, could presumably be reduced but not entirely prevented by the prophylactic use of exchange transfusion repeated as often as necessary, though the new threshold level of bilirubinaemia has yet to be determined.

Deafness afflicts 30% or more of survivors from kernicterus,[18] though haemolytic disease itself does not account for more than 3% of cases of congenital perceptive deafness.[19] The deafness is usually of high frequency type, similar to that occurring after prematurity or birth hypoxia, which raises the question of a common factor in pathogenesis. Haemolytic disease accounted for 15% of a large series of infants with obstructive jaundice,[20] but the prognosis is good, and permanent obstruction of the bile ducts does not occur, though rare cases of cirrhosis of the liver are reported. Congenital malformations and hepatolenticular degeneration are now known not to be connected with blood group incompatibility. In conclusion, it is heartening that 95% of the Armitage and Mollison series and 90% of Creak's series showed entirely normal mental and physical development on follow-up.

THE NUFFIELD FOUNDATION

The Nuffield Foundation, which was established nearly twelve years ago, in the middle of the war, has issued two progress reports.[1 2] One of them is technically an annual report, but the work supported by the Foundation is largely long-term and cannot be fitted into a single year, some of it not even into a five-year period. The annual report is really a report of what has been done during the last quinquennium and a prospectus of what it is proposed to do during the next. The other volume gives the list of the grants made over the last ten years, amounting to well over £4m. Research has been subsidized in many laboratories and university and other departments at home and overseas, the subjects ranging from nuclear physics to the effects of ageing, from dental health to soil science, from foetal physiology to the impact of television on children, from studies of the aborigines of Arnhem Land to the health services of the Aden Protectorate, from malnutrition of children in Uganda to the insect fauna of the game reserves of South Africa, and so on almost interminably.

The first grant of the Foundation in the medical field —£100,000 over ten years—resulted in the establishment of the Institute of Child Health of the University of London. Then industrial health departments were promoted in three universities in industrial areas of Great Britain, help being given to a total of £150,000. Dental health claimed attention, and a chair of oral medicine was set up in the University of Durham and research units in preventive dentistry in Leeds and Manchester. The Foundation has not concerned itself with particular diseases, with the exception of rheumatism; and on research in rheumatism, assisted by what is known as the Oliver Bird Fund, it has spent a total of £228,693 since the initiation of the Manchester centre in 1947. The care of the aged is another of the objects enjoined upon the Foundation by its trust deed. To the National Corporation for the Care of Old People it has contributed over half a million pounds in five years, and another half-million has been given by the Lord Mayor's National Air-raid Distress Fund. Finally, there is a large scheme of fellowships, scholarships, and bursaries, for residents both in the United Kingdom and in other parts of the British Commonwealth, to the number of several hundreds, about one-third of them in medicine. Seventy pages of this ten-year chronicle are devoted to a bibliography of published research attributed in whole or in part to the Foundation's grants and awards. Under the heading of research in the field of rheumatic diseases alone there are well over 100 entries, and that is only a selected list.

In the other volume, which is described as the ninth annual report, it is stated that during the last five years the Foundation has set aside nearly £400,000 for projects outside the United Kingdom. In the overseas parts of the Commonwealth private aid is "more keenly needed, sought, and valued" than in the United Kingdom, because in this country there is increasingly generous provision for scientific and medical research from public funds supplemented by industry. The Foundation's main concern during the next five years will be with the natural sciences (giving preference to biology), with continued support for research in rheumatism and some other medical projects, and with the social sciences. It will assist research and practical experiment in the employment of elderly people, and the various fellowship schemes will also be continued into the next quinquennium, but perhaps not all of them throughout the period. Some may be replaced by new schemes, for the Foundation is not committed to a rigid formula of beneficence. Indeed, apart from the scale of its benefactions, its chief characteristics seem to be its adaptability, imagination, and capacity for adventure—qualities also favoured by the great industrialist who provided this immense capital fund of £10m. for the advancement of health and of social well-being.

[1] *The Nuffield Foundation—Report on Grants, 1943–53*, 1954, London.
[2] *The Nuffield Foundation—Report for the Year ended March 31, 1954*, London.

THE GUT PROVOKED

"Diagnostic roentgenologists basically are organologists," says Friedman,[1] and in the interests of their search of the gut for growths, ulcers, polyps, and so on they usually do their best to soothe and console the patients committed to their rays. Friedman did exactly the opposite. By what he calls "superficial psychic probing" he provoked an emotional disturbance in patients under radiological examination, and claims thereby to have demonstrated the appearance of a "deficiency" or "motor dysfunction" pattern in the small intestine of persons in whom it was absent when they were in a state of composure. This is the pattern typically seen in pictures of the small intestine in cases of steatorrhoea, provided barium capable of flocculating is used. Friedman's patients were not suffering from steatorrhoea, and the illustrations of the small-intestine pattern before and after provocation are impressive. The main interest, however, of this study lies in its dual approach, the use of physical and psychological methods to study intestinal function.

A perusal of the work of Cannon[2] and the later studies of Wolf and Wolff[3,4] and their colleagues, aided perhaps by a little intelligent self-observation, must convince all but the most hard-boiled of organologists that the digestive apparatus may be profoundly upset in states of emotion. But whether these disturbances are of importance in the aetiology of so-called organic disease is a vexed question, for the technical difficulties of proving or disproving such a thesis seem at present almost as great as the philosophical ones in such questions as, Can mental events cause physical disease?

In the meantime the observation and measurement, when possible, of changes in function during emotional states is of value both for its own sake and to define more clearly the normal limits, thus to prevent these changes from being misinterpreted as evidence of structural disease. Friedman has shown that radiological methods can be used successfully for this purpose in the gut, but the danger of excessive irradiation must seriously limit the scope of the method. Colonic movement, for instance, has been studied in animals by means of serial *x*-ray pictures, and would appear to be an admirable subject for this kind of observation in man were it not for the excessive amount of irradiation necessary to obtain an adequate serial record of the movements of the colon.

Perhaps one of the most interesting aspects of this study is to illustrate how the old distinction between "organic" and "functional" in the sense of physically or mentally determined is breaking down. Is the disturbance which appears on *x*-ray plates as the "deficiency" or "motor dysfunction" pattern organic or functional? Perhaps the question does not make sense, and what we should ask in this, as in all changes of function in living persons, is: How far can this behaviour be explained by physical methods, and how far by psychological? The further applications of such a dual approach to gut function, whether by means of *x* rays or not, may well bring information of clinical value, and at the same time further illuminate gut–mind relationships.

[1] *Amer. J. Roentgenol.*, 1954, **72**, 367.
[2] Cannon, W. B., *Bodily Changes in Pain, Hunger, Fear and Rage.* New York. 1929.
[3] Wolf, S., and Wolff, H. G., *Human Gastric Function.* 1943.
[4] Grace, W. J., Wolf, S., and Wolff, H. G., *The Human Colon.* London. 1951.

UNEQUAL TRANSRADIANCY OF THE LUNGS

Chest radiographs showing one lung to be more translucent than the other are commonly seen when pulmonary distension has occurred to compensate for lobar or segmental collapse of a lung. A similar radiological picture is seen when partial bronchial occlusion has led to obstructive emphysema, and more rarely when an abnormality of the chest wall, such as loss of breast tissue following amputation, or congenital absence of the pectoral muscle, gives the illusion of abnormal translucency. Recently Macleod[1] drew attention to a group of patients whose radiographs showed abnormal transradiancy of one lung but in whom none of the usual causes were apparent. In nine such cases care had been taken to exclude abnormalities of the bronchi by bronchography and bronchoscopy. Most of the patients investigated suffered from increasing shortness of breath; cough was uncommon, but many of the patients were troubled by wheezing. Physical examination demonstrated diminished chest movement and reduced breath sounds on the affected side. Rhonchi, when present, were evenly distributed over both sides of the chest. Radiographs taken in inspiration showed increased translucency of one lung, the mediastinum being displaced towards the affected side in six of the patients. On expiration the difference in transradiancy was increased, and mediastinum shifted towards the normal side. Bronchograms showed that the finer bronchial radicles scarcely filled with oil and that there was almost no alveolar filling at all; there was, however, no abnormality in the calibre or distribution of the main bronchi.

These features made it clear that the patients were not suffering from obstructive emphysema, bullous emphysema, or cystic disease of the lung. In seeking an explanation for the underlying abnormality, only clinical evidence was available, since in no case had morbid anatomical or histological examination been possible. Macleod thought it unlikely that there was a congenital basis to these cases. He thought that some of them might be examples of unilateral emphysema, and measurements of the intrapleural pressures in one patient were in keeping with this diagnosis. The six cases in which the mediastinum was displaced to the diseased side were more difficult to explain. Here, Macleod thought the most likely explanation was that the affected lung had, during the time when growth was taking place, been affected by some disease which temporarily retarded its development. Later, when the primary disease had abated, the affected lung was stretched beyond its normal capacity in an effort to fill the hemithorax. It is quite conceivable that such a sequence of events could take place as a result of primary tuberculosis with transitory partial bronchial obstruction. The natural history of this disorder needs further observation before it is fully understood.

[1] *Thorax*, 1954, **9**, 147.

HEALTH OF SCHOOLCHILDREN

Since the end of the war children's mortality rates for infectious diseases have startlingly diminished, and even for diseases less susceptible to antibiotic attack they have shown a gratifying fall. The death rate per 100,000 for children aged 5–9 was, for infectious diseases (including tuberculosis), 22.7 in 1946 and only 6.5 in 1952. For children aged 10–14 the fall was from 19.6 to 4.9.[1] Rates for other causes combined fell to about two-thirds of their 1946 values. It is against this background that the Chief Medical Officer of the Ministry of Education can report[2] that in 1953 the health of schoolchildren was everywhere satisfactory. The general condition of the children examined at medical inspections was found to be "good" in just under 45%, "fair" in 53%, and "poor" in just over 2%. Diphtheria is now an uncommon disease owing to the immunization campaign. There were fewer outbreaks of food-poisoning, and probably no increase in the prevalence of rheumatic fever—possibly a fall. But in contrast to the great improvement in response to serious disease the C.M.O. has to record deterioration in other respects, though the diseases concerned are milder or relatively trivial. Perhaps most notable is the increase in dental caries. Indeed, "the figures of all areas show a substantial increase of caries in 1953 as compared with 1948." The most likely cause seems to be the almost incessant consumption of sweets, chocolate, and buns[3] that has become habitual to the children of far too many ignorant or over-indulgent parents. In spite of modern pesticides, over 300,000 children were found to be verminous, a figure that the report concedes to be disappointing even though it is the lowest so far recorded. No doubt education has a large part to play here, for in some sections of the population lice are looked upon as more or less normal commensals, and lousy parents provide a constant supply of fresh reserves as the lice infesting their children are wiped out. More and better co-operation between school doctors and general practitioners is a happy feature of the report, and by and large relations with hospitals seem to be cordial. But as Dr. C. W. W. Jeremiah, of Nottinghamshire, is quoted as recording: "Co-operation is a matter of personal relationship and cannot be dictated by 'powers that be,' be they national or parochial."

AGENE TREATMENT TO END

Last week the Minister of Food announced in Parliament[4]—some would think rather belatedly—that the agene treatment of flour is to end after December 31, 1955. Almost five years ago the Ministries of Health and Food concluded that the use of nitrogen trichloride (the agene process) for improving flour ought to be discontinued, and, as this *Journal* commented at the time,[5] "it is disquieting that not until over three years after a possible danger to health was shown to exist have steps been taken to combat it." Now that those steps have plodded on for another five years—and are to continue for almost twelve months more—the disquiet may at last give way to congratulation that the care of Parliament for its subjects' and of industry for its consumers' health has caught up with a scientific discovery made eight years ago. It was in 1946 that Sir Edward Mellanby reported in this *Journal*[6] that agenized bread is a cause of canine hysteria, and a year later he found that it could cause the same symptoms in ferrets.[7] In 1950 Mellanby, together with his colleagues P. N. Campbell and T. S. Work, isolated a crystalline toxic factor from agenized flour which on administration to ferrets caused severe epileptiform fits.[8] It was apparently the same as a product obtained independently by a team working in the laboratory of the British Flour-Millers Association. Mellanby *et al.* emphasized that the poison was cumulative in effect: a fatal toxic reaction was produced by feeding to ferrets over five to ten days doses that were only a fraction of those that caused an immediate toxic response.

While animal experiments showed that the product of agenization is exceedingly poisonous to some animals, no satisfactory evidence has been adduced that it is harmful to man. The presumption is nevertheless reasonable that a substance so poisonous to some mammals ought not to be incorporated in human food, especially in such a staple food as bread. Popular taste may well demand "improved" flour, and limits of storage space necessitate it, but the replacement of agenization by another process can only be welcomed. What the best method will be is still under investigation. The chief chemist of J. Lyons and Co. recently referred to some of them,[9] and in particular to a new method of mixing the dough so as to oxidize the flour by means of aeration. To anyone who has a healthy scepticism about adding chemicals to food this line of research would seem to be well worth following up.

EMERGENCIES IN GENERAL PRACTICE

The first of a new series of articles in the Refresher Course for General Practitioners appears in this issue at page 39. The articles will all be concerned with emergencies in general practice—medical, surgical, obstetric, and psychiatric—and will range in subject from such common disorders as cardiac asthma, discussed this week by Dr. J. G. M. Hamilton, to the accidental swallowing of poisons by children, and from post-partum maternal collapse to acute lumbago and sciatica. Articles previously printed in the Refresher Course series have proved popular in book form, two volumes having so far been published and the third, *Clinical Pathology in General Practice*, being in the course of preparation. The aim of the new series on emergencies is to provide reliable, up-to-date, and practical advice, bearing in mind the nature of domiciliary practice and the special problems of the general practitioner.

[1] Martin, W. J., *J. Hyg. (Lond.)*, 1954, **52**, 417.
[2] *The Health of the School Child: Report of the Chief Medical Officer of the Ministry of Education* 1952–3. H.M.S.O. 5s.
[3] Dental Health of Children: Memorandum by the Child Dental Health Committee of the British Dental Association, *Brit. dent. J.*, 1954, **96**, 199.
[4] See *Journal*, p. 50.
[5] *British Medical Journal*, 1950, **1**, 295.
[6] Ibid., 1946, **2**, 885.
[7] Ibid., 1947, **2**, 288.
[8] *Nature (Lond.)*, 1950, **165**, 345.
[9] *British Medical Journal*, 1954, **2**, 1410.

EMERGENCIES IN GENERAL PRACTICE

CARDIAC ASTHMA

BY

J. G. M. HAMILTON, M.B., F.R.C.P.Ed.

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The term "cardiac asthma" is applicable to a clinical syndrome—paroxysmal dyspnoea due to heart failure—with a range of severity extending from urgent acute pulmonary oedema to transient nightly attacks of cough, wheezing, and breathlessness, though used by some to refer only to the latter. Although such paroxysms sometimes occur in the presence of mitral stenosis, they are most commonly an expression of left ventricular failure in hypertensive, coronary, or aortic valvular disease. A sudden attack of intense dyspnoea is not infrequently caused by acute myocardial infarction, and the occasional absence of the characteristic pain or the overshadowing of pain by the intense dyspnoea may result in the basic lesion being missed.

Clinical Picture

The patient is most commonly in the second half of life and may or may not be known to suffer from hypertension or coronary disease. He is wakened from sleep, or roused from quiet, recumbent rest, by cough and breathlessness which may rapidly reach urgent severity so that he is obliged to sit up gasping for breath and hardly able to speak. Soon some frothy, watery sputum appears, becoming in the most severe attacks profuse and pink through blood-staining. Wheezing may be prominent. The patient's distress is often accentuated by the occurrence of Cheyne–Stokes periodic respiration. During the phases of apnoea he may fall back on his pillows and close his eyes in thankful comfort, only to be roused in a few seconds by terrifying distress as the phase of hyperpnoea develops. The tantalizing repetition of the cycle may be almost unbearable.

In less severe attacks of breathlessness with cough and wheezing the intensity of the symptoms may decline spontaneously after the patient has been sitting up for 20 to 30 minutes and he may return to sleep without medication. While a severe attack is sufficiently alarming for medical help to be summoned, in the case of the lesser attack the patient commonly consults his doctor only after several disturbed nights. He may be free from breathlessness by day or admit only to some effort distress if questioned. The described picture of cough, wheezing, and breathlessness all too often leads to an erroneous diagnosis of bronchitis or bronchial asthma and the prescription of an ineffectual expectorant or linctus, or the administration of isoprenaline or adrenaline.

The patient taken urgently ill in the night is seen to be sweating and cyanosed, with cold extremities. The heart is accelerated, while the rhythm, commonly regular, may be disturbed by extrasystoles or auricular fibrillation. The blood pressure is usually elevated, often grossly, pulsus alternans being often observable, but may have fallen from a previously hypertensive level to a normal or low level either because of the severity of the heart failure or because the attack has been precipitated by the occurrence of acute myocardial infarction. A complaint of gripping, crushing, or burning retrosternal pain, severe or slight, prior to or coincident with dyspnoea may draw attention to this possibility, which is of more than academic interest, even in the middle of the night, for in several respects treatment should be varied according to whether or not infarction has or seems likely to have taken place. Cardiac enlargement is almost invariably to be found and should be looked for by palpation of the apical impulse, for this provides the necessary conclusive evidence of the existence of organic cardiac disease, and, in the presence of a normal or low blood pressure and the absence of aortic valvular disease, establishes that hypertensive heart disease underlies the condition. Further valuable evidence of heart failure is provided by the finding on auscultation at the apex of triple or gallop rhythm. The neck veins are often somewhat engorged, but oedema of ankles or sacrum is generally absent, since attacks of cardiac asthma usually antedate the appearance of right heart failure and indeed may cease to occur when right ventricular failure supervenes.

Although respiration may be noisy with wheezing rhonchi, crepitations are to be heard over both lungs, in severe cases in all areas to such an extent and in such profusion that the lungs seem waterlogged, in less severe cases predominantly at the lung bases.

In the case of the patient examined between milder attacks similar evidence of heart disease will be found—hypertension, cardiac enlargement, and basal crepitations, although these last may be scanty at such a time.

The need to elicit these signs of heart disease has been stressed, since there is no little chance that hasty consideration of the symptoms of cough, wheezing, and dyspnoea, and the finding of harsh respiration with rhonchi and crepitations, may focus attention on the respiratory tract and lead to a diagnosis of bronchitis.

Paroxysmal dyspnoea of this type—cardiac asthma—except in the instances in which it is actually precipitated by myocardial infarction or paroxysmal tachycardia, occurs for the most part at night before or during sleep. It may be the first clear manifestation of heart failure in the hypertensive subject, though it often comes after a decline in effort tolerance has set in. Cardiac asthma, however, seldom develops for the first time in patients already under adequate treatment for hypertensive heart failure unless it is due to the occurrence of some complication such as infarction or a paroxysmal arrhythmia. Severe nocturnal attacks may follow unduly vigorous exertion during the preceding day, or an injudiciously large meal in the evening, or high excitement, including intercourse.

Cause of the Dyspnoea

The symptoms of cardiac asthma are immediately due to pulmonary engorgement. Quiet rest in bed with its decline in physical effort and reduction in metabolism is associated with reabsorption of tissue oedema fluid, even where this has not been manifest, and therefore with an increase in circulating blood volume. The recumbent posture results, owing to the removal of the influence of gravity, in an increased venous return to the right side of the heart which, through increased venous filling pressure, is a stimulus to increased ventricular output. In the presence of hypertension or aortic valvular disease or coronary sclerosis, whose main detrimental influence is upon the left ventricle, this chamber, beginning to reach the stage of dilatation and failure, may be less able to respond to such a demand for greater effort and output than the less directly burdened right ventricle. The vascular bed between these chambers—that is, the pulmonary capillaries and venules—is congested therefore to accommodate the excess of the right ventricle's output over that of the left, tiny though that excess may be at each stroke.

Pulmonary congestion and the resulting transudation of oedema fluid into the alveolar walls and cavities progressively reduce the vital capacity, while the emergence of this fluid on to the sensitive bronchial epithelium touches off

the cough reflex. Either the dyspnoea of reduced vital capacity and stiff congested lungs or the cough wakens the patient. The intense effort of the dyspnoea and cough constitutes very vigorous exercise which itself aggravates the cardiac embarrassment. An additional factor operating in some cases is general peripheral arteriolar constriction. The increase in arterial pressure which this causes throws a sudden additional load on a left ventricle already prejudiced by established hypertensive disease, coronary ischaemia, or both, and precipitates left ventricular failure. In such cases the blood pressure is found to be elevated well beyond what is known to be that patient's customary level.

In the mild or moderate case the assumption of the erect posture when the patient sits up in bed, or better still in a chair with his feet on the floor, may break the vicious cycle of pulmonary engorgement→dyspnoea and cough→exercise→further pulmonary engorgement→worse dyspnoea, by allowing a reduction in venous return so that the labouring left ventricle can gradually overtake its debt and clear the pulmonary congestion below the critical level. Then the attack may end spontaneously. In the worst cases the vicious cycle progresses and the patient dies, drowned by his own pulmonary transudate.

Partial ischaemia and anoxia of the respiratory centre resulting from diminished arterial output from the overloaded left ventricle, combined probably with a measure of cerebral vascular stenosis, especially in the elderly, is the cause of Cheyne–Stokes respiration in cardiac asthma. It is not to be attributed to uraemia in the circumstances under discussion.

Immediate Treatment

Treatment of cardiac asthma resolves itself into the handling of the emergency and the long-term management of the underlying disorder with the object of rendering less likely the repetition of such disturbing and dangerous crises which are almost certain to recur if the crisis is taken on its own and sustained treatment neglected.

In the emergency morphine should be administered immediately the diagnosis of *cardiac* asthma has been made. The object here is twofold : first, to reduce the sensitivity of the cough reflex and of those respiratory reflexes having their origins in the stiffened congested lungs, whereby the intensity of the effort of coughing and breathing may be diminished, and, secondly, to calm the apprehension, even panic, and the restlessness which a severe attack of cardiac asthma causes in even the most phlegmatic person. With these effects achieved venous filling pressure on the right heart and therefore right ventricular output are reduced and the left ventricle given an opportunity of partially clearing the congested pulmonary vascular bed. The effects must be achieved with the maximum certainty and without delay, since any severe attack of this condition must be regarded as critical and immediately endangering life. For a patient of average or over-average weight morphine sulphate, ¼ gr. (15 mg.), should be injected intravenously and the same quantity subcutaneously. In the presence of acute heart failure absorption of the whole dose from a subcutaneous injection is undesirably slow, and if intravenous injection is for any reason impracticable, half of the total dose of ½ gr. (30 mg.) should be given by intramuscular injection. In the case of patients of under-average weight the total dose can be reduced to ⅓ gr. (20 mg.), but again half of this dose should be given intravenously. When the attack is of only moderate severity the ordinary subcutaneous injection of morphine, ¼ gr. (15 mg.), may suffice.

Many practitioners combine a dose of atropine sulphate with the subcutaneous injection of morphine or even give atropine alone. This habit is based upon a misconception that the excessive fluid in the respiratory passages is due to excessive bronchial secretion. However, the fluid is in reality transuded oedema fluid whose production cannot be controlled by paralysing by atropine the bronchial secretory glands. If atropine has any action in cardiac asthma it is by depressing vagus-mediated reflexes involved in the production of dyspnoea. Atropine is better omitted altogether, since the dryness of the mouth which it induces makes the patient demand fluid to drink which he would be better without.

Although Cheyne–Stokes respiration, if present, may be expected to be aggravated by the injection of a large dose of morphine, this must not deter the physician from giving morphine in an adequate dose. The distressing dyspnoea (which is what immediately matters) of the hyperpnoeic phases will be suppressed. The periodicity of the respiration, however, can usually be rapidly relieved by an intravenous injection of aminophylline, 0.5 g. Such an injection should be given in all severe or moderate cases, even in the absence of Cheyne–Stokes breathing, for its vasodilator and bronchodilator action.

The patient should be propped securely on a high bank of pillows or made comfortable in an easy chair.

Other Measures

The question of the administration of digitalis or digitalis glucosides requires rather more careful consideration in each case. When the patient is known not to have been taking digitalis or digoxin for at least a week before the attack, and when there is no reason to suppose that acute myocardial infarction has occurred, digoxin, 1 mg., should be injected intravenously directly after the administration of morphine. It may be combined with the injection of aminophylline. When the patient is known to be fully under the influence of an adequate maintenance dose of digitalis or digoxin already, it is wise to assume at the time of the acute episode that the attack is due to the occurrence of myocardial infarction and withhold the intravenous injection of digoxin. Such should also be avoided, even in the undigitalized patient, when the features of the case strongly suggest that an infarction has precipitated the crisis. The risk of inducing paroxysmal ventricular tachycardia or even fatal ventricular fibrillation is not negligible. Reliance should then be placed on intravenous morphine and aminophylline for the emergency and digoxin, 0.5 mg., given by mouth as the starting point of a more gradual process of digitalization.

Where there is a strong clinical indication that acute myocardial infarction has taken place, early and rapid anticoagulant therapy with heparin may prevent an immediate extension of the infarct, with serious and perhaps fatal results. Heparin, 10,000–15,000 units (100–150 mg.) according to body weight, should be given intravenously and repeated six hours later, after which it can be decided whether a full course of anticoagulant therapy is to be undertaken.

If oxygen is readily obtainable it should be used. It is desirable that a flow-meter should be employed and a flow of 6–8 litres a minute administered by double nasal catheter or by a light plastic nose-and-mouth mask. The patient should be allowed to go to sleep with this in place, but naked lights should not be permitted near the oxygen cylinder or the mask.

If in a severe case dyspnoea does not subside within 10 to 30 minutes of the injection of morphine, *venous* tourniquets should be applied on the proximal parts of all four limbs to reduce venous return. They should be tight enough to hamper the venous outflow from the limbs but not tight enough to impede the arterial inflow. After about an hour the tourniquets should be *slowly released one at a time* to avoid a sudden augmentation of venous return which would again overload the recovering heart.

Rapid venesection of one pint (570 ml.) through a wide-bore needle, or by simply slashing a vein, may be usefully employed instead of the above "bloodless phlebotomy," especially in plethoric patients.

When a striking rise in blood pressure is observed, suggesting a hypertensive crisis, the use by intravenous injection of powerful vasodilators and hypotensive agents such as tolazoline and the methonium compounds has much to

commend it on theoretical grounds. In patients suffering from severe acute left ventricular failure due to hypertension the rapid reduction of an extravagantly high blood pressure may so reduce the load on the failed left ventricle as to contribute importantly to the interruption of the vicious cycle. Beneficial effect has been obtained in many such cases, but the use of methonium compounds by intravenous injection is fraught with risks and is better avoided except by those with experience of their use in this and other fields.

Long-term Management

On the morning after the severe attack the long-term measures should begin. Arrangements should be made for the patient to be confined to bed, well propped up at night, or to bed and a chair in his room according to the severity of the condition, in the "uncomplicated" case for about two weeks, but of course for longer when myocardial infarction has been diagnosed. Mersalyl, 2 ml., should be given by intramuscular injection, to be repeated two days later, twice weekly for two to three weeks, and weekly thereafter, even though manifest peripheral oedema be absent. A more cautious approach to the use of full doses of a mercurial diuretic—for example, mersalyl, 0.5 ml. for the first dose and 1 ml. for the second—is indicated where previous symptoms of prostatic hypertrophy suggest the possibility of acute retention being precipitated by the sudden massive diuresis of the full dose. The process of thorough digitalization should be continued by ordering, for example, digoxin, 0.25 mg. thrice daily by mouth. Later, when a full digitalis effect has been achieved, as indicated by the appearance of anorexia and squeamishness, the dose should be adjusted, after 24 to 48 hours' "desaturation-holiday," to one suitable to maintain an optimum digitalis effect for the rest of the patient's life. The occurrence of cardiac asthma should be the signal in the untreated patient for the institution of the "digitalis life."

Moderation of salt intake should be arranged by the prohibition of salty foods and added salt, the limitation of salt in cooking, and the avoidance of sodium-containing medicines. If severe sodium restriction can be achieved and if a mercurial diuretic is regularly given the patient can be permitted a normal fluid intake.

For three or four nights after the acute attack of cardiac asthma morphine, ¼ gr. (15 mg.), should be given subcutaneously and for a week thereafter an opium-containing hypnotic given by mouth, such as:

Chloral hydrate	gr. 20 (1.3 g.)
Tincture of opium	min. 20 (1.2 ml.)
Liquid extract of liquorice	min. 10 (0.6 ml.)
Water	to fl. dr. 2 (7 ml.)

It is important that until the full effect of digoxin, mersalyl, and rest has been obtained the patient be protected by opium against a recurrence of the nocturnal crisis.

In the case of the patient complaining of recurrent milder attacks of cardiac asthma at night an opium-containing hypnotic is indicated for seven to ten nights. For this purpose a mixture like the above, but with 30 gr. (2 g.) of chloral hydrate and 30 min. (1.8 ml.) of tincture of opium, is suitable, half the dose being given in the last few nights of the period or for a few further nights if required. He should be advised to use extra pillows and to avoid slipping down to a low position if he can. While the patient is thus protected against the nocturnal attacks he should be brought fully under the influence of digoxin in the usual way and a permanent maintenance dose established. Again, even in the absence of manifest oedema, intramuscular injections of mersalyl, 2 ml., should be given in the morning twice a week for two or three weeks, and weekly thereafter.

The other measures called for in the treatment of cardiac failure—salt limitation and modification of habits in respect of physical and mental effort—will if necessary be inaugurated at the same time. In the milder cases under consideration, unless there are manifest features of chronic heart failure or frank evidence of serious coronary disease, it is seldom necessary to confine the patient to bed for more than perhaps a day or two, though a week or two off work with modified rest about the house is desirable while the full effect of treatment is being attained.

Conclusion

There must be few acute disorders in the non-surgical realms of adult medicine in which treatment is so immediately rewarding as in cardiac asthma. The utter relief of the acutely dyspnoeic patient, or the achievement of nights blessed by sound sleep undisturbed by attacks of wheezing breathlessness in the less severely ill patient, does, for me at any rate, amply reward the night-call, the bloody mess of the venesection, or the early morning injection of mersalyl. However, it cannot be too often or too forcibly emphasized that the foundation of the emergency treatment of this serious and dangerous disorder is its recognition as heart failure and not bronchitis, and the use of *morphine*, aminophylline, and digoxin, and of its prevention the permanent treatment of hypertensive and coronary heart disease with digoxin, mersalyl, and salt restriction.

Next article on Emergencies in General Practice.—"Respiratory Insufficiency in Poliomyelitis and Other Diseases," by Dr. W. Ritchie Russell.

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THE SWEEP-FREQUENCY METHOD OF MAKING SCREENING TESTS OF THE HEARING OF SCHOOLCHILDREN

STATEMENT PREPARED BY PROFESSOR A. W. G. EWING AT THE REQUEST OF THE M.R.C. COMMITTEE ON THE EDUCATIONAL TREATMENT OF DEAFNESS*

In a recent investigation it was found that approximately 70% of pupils in English schools for the deaf and partially deaf have suffered from their handicap since before the age of 2 years. From the Ministry of Education's returns and from the results of recent surveys it seems that nearly all deaf children are admitted to special schools by the age of 7 years, but that the ascertainment of partial deafness as a cause of educational retardation is at present less effective among children under 7 than among those who are older.

The sweep-frequency technique has been shown to be a reliable and efficient method of making screening tests of the hearing of 5- and 6-year-old schoolchildren. A pure-tone audiometer is used, and, when the range of frequencies that are used is extended from 250 to 6,000 cycles per second, early symptoms both of conductive and of perceptive deafness can be detected. It has been established that a sweep check over this range of frequencies at 15 decibels above normal threshold of detectability is more thorough and reliable as a screening test than the use of word material (as in the test by gramophone audiometer), the loudness of which cannot be monitored satisfactorily for this purpose. The fading-intensities, called-number test has been shown to be sometimes ineffective for the detection of high-tone deafness.

*Sir Frederic C. Bartlett (chairman), Mr. T. E. Cawthorne, Professor A. W. G. Ewing, Mrs. I. R. Ewing, Dr. D. B. Fry, Mr. C. S. Hallpike, Mr. D. Lloyd James, Mr. J. Lumsden, the late Mr. J. Spalding, Mr. A. B. Swayne, and Professor R. C. Oldfield (secretary).

Audiometricians who have been trained to work with young children are able to make a sweep check of auditory acuity, as an individual test, at a rate of three minutes per child. It has been shown that extraneous noise in a room in which a screening test of hearing is to be made should not exceed about 30 decibels. The sweep-frequency test can be made in a small room. When it is necessary to make screening tests of children's hearing in school buildings it is much less difficult to find small rooms that are quiet, in schools, than large rooms.

The sweep-frequency test can be made immediately or soon after children are first admitted to school, whereas the gramophone audiometer test, as normally administered, is a group test, in which children are required to write down numbers that they hear. It cannot therefore be used with children younger than 8 years. The sweep-frequency test is very valuable in screening out children who are likely to need hearing-aids and special educational treatment, before they have been exposed to the risk of educational subnormality as a result of defective hearing.

A comparative study of the relative reliability of the sweep-frequency and gramophone audiometer tests, when both were administered to 6-year-old children individually, showed the sweep-frequency test to be superior. The called numbers that are used in the gramophone audiometer test involve a constrained context and are therefore often recognizable by children suffering from loss of hearing to a significant degree.

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Nova et Vetera

BLEEDING, CUPPING, AND PURGING

When Roderick Random, newly qualified, was seeking a position as an assistant to a well-established practitioner, he was faced with the pertinent question: "Can you bleed and give a clyster, spread a plaster, and prepare a potion?" From the beginning of history these three arts—together with that of providing more active counter-irritation by the seton or by the cautery—had formed the basis of the doctor's technique. In his recently published FitzPatrick Lectures for 1950–1* Dr. William Brockbank discusses authoritatively as much of the vast history of these subjects as can be fitted into the compass of a volume of reasonable size, and he has added an interesting summary of the history of intravenous injection. There is no similar work which covers the same field.

The enema, or clyster—for though philologically distinct the words are practically synonymous—has a very ancient history. Pliny appears to have been the originator of the myth that the black stork of Egypt used its long beak to give itself an enema of Nile water. Mediaeval illustrations show the bird carrying out the operation. But in fact the bird is probably only taking oil from its preen gland to preen its feathers. No modern observer has ever seen a bird acting as in Pliny's account.

The antiquity of the enema is shown by its distribution among primitive peoples. For example, the Eskimos use a portion of the gut of a seal, into one end of which a bone nozzle is bound. The simple enema tube was used by the Aztecs of Mexico and by the Maya of Yucatan. Other ancient races, such as the Peruvians, fitted a bulb to the enema tube, the bulb being often formed by the bladder of an animal. Both in Africa and in America gourds are commonly used as enemata at the present day, and a reed is frequently fitted to the narrow end of the gourd.

Excelsior

From the very earliest times the use of the enema has been cursed by an erroneous motto—*Excelsior*—on the part of its adherents. The Babylonians tried to reach the whole of the intestinal tract by giving their nauseous mixtures both by mouth and by the rectum at the same time. The ileo-caecal valve was not known until 1588, and much damage must have been caused by those appliances which attempted to drive the injection round the bowel by force. The most powerful apparatus of this type was finally invented by Equisier in 1848. In this apparatus the fluid was injected suddenly by means of a powerful spring.

In general all practitioners up to the mediaeval period used what might be termed "rectal polypharmacy," and many an inflamed colon must have been violently irritated by the drugs employed. Avicenna was the first to use soap and water as the common basis. John Arderne, who practised surgery at Newark-on-Trent in the second half of the fourteenth century, wrote a celebrated treatise on fistula, and he gave a description and illustration of his enema apparatus. Arderne used a swine's bladder to inject the fluid. (There is, incidentally, no justification for calling him, as Dr. Brockbank does, John of Arderne.) The piston syringe as a means of propelling the injection is shown in a fifteenth-century carved-wood apothecary's sign which was presented to the Museum at Bruges in 1880; but the syringe may have been in use earlier, since such a method is employed by certain primitive races, notably the Catawba Indians of North Carolina.

At certain periods patients appear to have been shy of allowing themselves to be treated by this method, while at other times they were just the opposite. Ambroise Paré seems to have been the first to introduce an enema syringe by using which the patient could administer the enema to himself. De Graaf and others invented similar types of apparatus, and in the eighteenth century the enema stool became popular. Molière and the Roi-Soleil unintentionally popularized the use of the enema, and from that time onwards it appeared in cartoons and on the canvases of the great artists. In 1695 Dekkers published a practical manual at Leyden, and in this he devoted nearly 100 pages to the use of the enema. In 1480 Louis XI, suffering from apoplexy, was relieved by an enema, and from then on he had his pet dogs treated by this method. Early in the nineteenth century an engraving was published showing a pet dog receiving an enema under the supervision of learned physicians. Satirists such as Rowlandson and Gillray frequently introduced the enema in their political cartoons. The rubber bulb attached to an enema tube seems to have been used in the Amazon region from early times. It was brought to Europe by the Portuguese shortly after 1740, and its use spread widely. Higginson invented his syringe about 1850.

Cupping Horns

Cupping has also an ancient history. The ends of animal horns are still used by primitive races, and in the Wellcome Collection there is the equipment of the personal physician of Cetewayo, the Zulu chief. It contains no fewer than thirteen such cupping horns. Dry cupping is performed by inverting over the skin a glass vessel which has been exhausted by a flame. It is an interesting and harmless experiment to carry out this simple operation on oneself. I have never seen the method used on a patient,

*William Brockbank, M.A., M.D., F.R.C.P.: *Ancient Therapeutic Arts. The FitzPatrick Lectures delivered in 1950 and 1951 at the Royal College of Physicians.* London: William Heinemann Medical Books, Ltd., 1954. Pp. 162. Price 25s. net.

but as a student its use was demonstrated to me as a method which had practically died out. Dr. Brockbank also says he has never seen a patient cupped; but he quotes very appositely George Orwell's description of his treatment for pneumonia in a Paris hospital in 1929—six cupping glasses, followed by scarification of the blisters, reapplication of the cups, and then a mustard poultice. This was really wet cupping, in which the aim was to draw blood into the evacuated cups. Paré introduced the mechanical scarifier, a devilish instrument which produced at once up to eighteen incisions within the area of the cup. These scarifiers were used until well into the nineteenth century. In some instruments the movement of the blades is so rapid that it is quite invisible.

Dr. Brockbank deals with the subject of leeching less fully than one might have expected. He might have said something about the bloody war which raged in France during the 1830's. On the one side Broussais, on the other P. C. A. Louis. In nine years (1824–33) the teaching of Broussais increased the number of leeches used in France from 3 to 40 million annually. It was at this period that a physician at the Val-de-Grâce was in the habit, when he arrived at the hospital for his visit, of asking how many patients had been admitted. He thereupon ordered 30 leeches each before he had examined them. There is a well-known satire which shows Broussais ordering a further ninety leeches for a moribund patient—"and continue the diet." Dr. Brockbank gives a most interesting account of the variations in the number of leeches used in the Manchester Infirmary between 1782 and 1882. These variations correspond with those described by Marshall Hall at the General Hospital, Nottingham.

On the subject of counter-irritation by other means Dr. Brockbank is very interesting. Cantharides as a blistering agent is of course of great antiquity, and, as the "fly-blister," was used in the early years of the twentieth century, and may indeed still be used. The seton is essentially a cord of silk or cotton inserted through the soft parts and left to drain away the resulting pus for a period of a year or more. Very frightening were the instruments for the insertion of setons invented by Paré, Glandorp, Bartisch, and others. An issue, or fontanelle, was a pea or a metal cylinder inserted in the superficial tissues to produce suppuration. It disappeared from practice early in the nineteenth century, and the seton a few decades later. A moxa was a cone of *Artemisia moxa* which was applied to the skin and slowly burned to produce a blister. This Chinese method of counter-irritation came to Europe in the seventeenth century and had been discarded before the middle of the eighteenth.

Dr. Brockbank's very readable and valuable book would be enhanced by the addition of an index. It is well produced—though the style of production may be rather modern to suit everyone. Some may still think that the best place for a caption is below its illustration and not on the opposite page. In this, as in some other "modern" books, the "title-page" can no longer be termed such, for it has been shorn of one-third of the title of the book. The erring word has wandered over to the reverse of the page traditionally known as the "half-title." The bibliographer will have to revise his terminology.

E. ASHWORTH UNDERWOOD.

A study group set up by the World Health Organization to consider the identification and classification of the snail vectors of bilharzia found that confusion existed not only in the nomenclature of the species but even in the delimitation of the genera and the family to which snails belong. It was therefore unanimously agreed that the family name should be "Planorbidae" and that the intermediate hosts should be placed in the two subfamilies "Planorbinae" and "Bulininae."—*Wld Hlth Org. techn. Rep. Ser.* No. 90, 1954, Geneva.

Preparations and Appliances

A FOOT EXERCISER FOR USE WITH A THOMAS SPLINT AND A TULLOCH BROWN U-LOOP

Dr. R. C. LEMON, accident and orthopaedic registrar, Royal Buckinghamshire Hospital, writes: All leg splints to-day have attachments for the prevention of foot-drop. The two exercisers in use at this hospital were designed not only to prevent foot-drop but also to enable the patient to plantar-flex his foot actively and against a spring resistance. They are easy to make, are simple in construction, and have been a great aid in keeping up the tone of the patient's leg muscles. They have been used in fractures, and in cases of arthroplasty of the hip in which traction has been deemed advisable by means of a Tulloch Brown U-loop. Both exercisers are basically the same in construction. The first can be used on any Thomas splint or its Pearson attachment (Figs. 1 and 2). It consists of a foot-rest, in this case made of aluminium, which can be adjusted up or down by means of a screw-bolt so that its curved upper end is at the level of the metatarsal heads. The foot-rest is attached to an axle which clamps to the side bars of the splint, the axle itself having a telescopic device so that it can be adjusted to any width of the splint (Fig. 2). The resistance is obtained by a spring attaching to the bottom of the foot-rest and to a clamp which can be screwed to either side bar of the splint at any required distance from the foot-rest to increase or diminish the resistance against which the foot has to work.

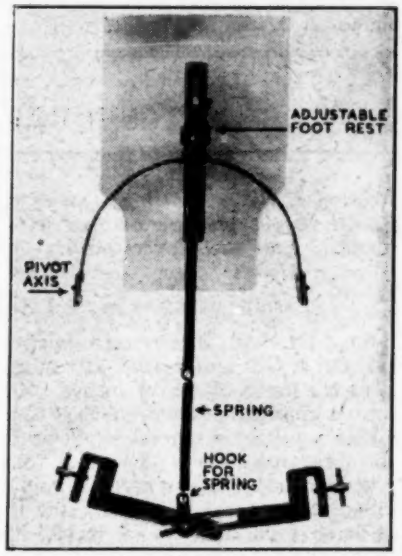


FIG. 1.—Tulloch Brown U-loop exerciser. The side bars of the splint are seen in the bottom of the picture.

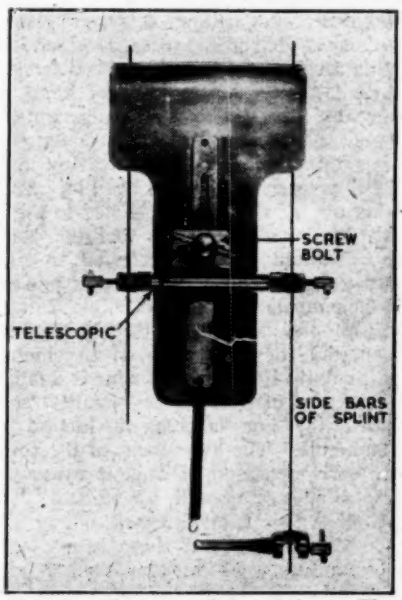


FIG. 2.—Thomas splint exerciser. The black lines indicate the side bars of the splint.

In the case of the exerciser for the Tulloch Brown U-loop, advantage has been taken of the Nissen foot-plate already provided with the splint. The foot-rest has been made adjustable up and down by means of a screw-bolt again.

Mobility is attained by cutting off the flanges on the side pieces. The attachment for the spring is made by fixing a rigid steel bar vertically downwards from the foot-rest (Fig. 1).

The exercisers are placed against the foot and the spring tension is adjusted so that when at rest the foot is held at 90 degrees. Plantar flexion can then be carried out with the greatest of ease.

A thick woollen bed-sock is used on the foot to prevent any sores developing and to facilitate movement of the foot against the foot-exerciser. Padding attached to the foot-exerciser itself interferes with the movement.

I am indebted to Mr. G. Platt for his helpful criticism and advice; to Mr. Dudley, who made the exerciser to my design; and to Mr. Mason for the photographs.

Correspondence

Because of the present high cost of producing the Journal, and the great pressure on our space, correspondents are asked to keep their letters short.

Changes in the Mental Health Services

SIR,—Dr. J. B. S. Lewis (*Journal*, December 4, p. 1354) and Dr. J. Gibson (*Journal*, December 18, p. 1484) strongly advocate the abolition of mental observation wards, but a mental observation ward attached to a general hospital and administered by a general physician can do valuable work, for these reasons: (1) Facilities for laboratory tests and consultant medical and surgical opinions being more easily obtained at a general hospital, the investigation and treatment of physical causes for mental illness can conveniently be carried out there. In my own observation ward a large number of people have been admitted as mental cases when their illness was entirely due to physical causes such as pneumonia, empyema, meningitis, cerebral tumour, G.P.I., bromide intoxication, hypoglycaemia narcolepsy, myxoedema, Addison's disease, Simmonds's disease, and many other conditions.[1] The early detection and treatment of these organic cases is surely of importance and more easily done by a man who is in daily contact with acute medical work. (2) In a general hospital many patients with medical or surgical illnesses may develop mental symptoms—the delirious pneumonias, the head injuries, and others. If there is an observation ward attached to the hospital these people can continue treatment under the doctors that know their case without being transferred to mental hospital. It is also most beneficial to the other patients in a general ward if their mentally disturbed fellows can be temporarily moved out of the general ward. (3) The unfortunate compartmentation of medical from psychiatric work is lessened, particularly if an appointment is established where one man has charge of both general medical and mental observation cases. (4) For teaching a mental observation ward is invaluable. The assessment of the acute psychotic case and the early detection of organic causes for mental illness are just what are needed by students. (5) There are many people with mental illnesses that only last a few days, and it is an advantage if these people can be managed in an observation ward without their having to go to mental hospital. (6) Where the assessment of a suspected case of mental illness is difficult, it is far easier to judge the patient if he is under observation for a few days rather than decide at a short interview in his home. A fair decision as to his sanity is hence more easily made when the question of certification arises, and for other patients the choice of voluntary status, temporary order, or discharge can be more wisely made.

For all these reasons I think that observation wards ought to continue and that the L.C.C. are right in recommending this to the Royal Commission. To argue, like Drs. Lewis and Gibson, that they should be abolished because some of them are overcrowded or badly staffed is not very sensible; some general hospitals are overcrowded and badly staffed, but one could not reasonably suggest abolishing all the general hospitals in the country for that reason. It is more logical to suggest that the staffing and equipment of observation wards should be improved where it is inadequate.

Observation wards are not always the places of noisy, nightmare horror your correspondents suggest. Often after a patient with delirium from pneumonia has been transferred to the mental observation ward from the general side I have, when he has begun to recover, said, "You can go back to the other side now," to be told, "I think I'd rather stay here, doctor—it's so much quieter."—I am, etc.,

London, N.W.10. RICHARD ASHER.

REFERENCE

[1] Asher, R. (1954). *Trans. med. Soc. Lond.*, 70, 93.

Premedication of Children for Tonsillectomy

SIR,—The summary of Dr. Christine M. Rendell's article (*Journal*, December 11, 1954, p. 1397) indicated that in methylpentynol had been found an almost ideal drug for pre-operative sedation of children prior to tonsillectomy. This appraisal is not, I contend, justified by the text, with the findings of which I am largely in agreement, though not with Dr. Rendell's evaluation of them. In a smaller series than Dr. Rendell's a clinical impression has been gained by me that methylpentynol is in fact a very unsatisfactory pre-operative sedative.

Everyone would agree that some premedication is helpful in allaying fear, and in my series it was entirely "verbal" and carried out by the ward sister or anaesthetist, the only pre-operative drug used being atropine 1/100 gr. (0.65 mg.) prior to guillotine tonsillectomy. Our experience indicates that with this preparation alone the apprehensive or frightened child is rarely encountered—rather less often than once in ten cases. At the present time an investigation is under way to test this impression on a scientific basis. In the light of this clinical impression, then, Dr. Rendell's claim for the success of methylpentynol is open to doubt, and is rather weaker than for cases where only atropine was used. Methylpentynol did not rob the procedure of psychic trauma; Dr. Rendell admits that an inquiry after the children's discharge established that "many . . . retained a vivid and unpleasant memory" of the induction despite the statement that at the time they were "peculiarly happy and co-operative." It is hard to reconcile these two observations, since surely distaste on recollection of the event must imply the same feelings at the time. I cannot endorse Dr. Rendell's assertion that children find the "oblivon elixir" pleasant to take. All of my series were questioned immediately prior to induction, at a time when a favourable reply might have been expected. Only two in fifty-six children admitted liking the preparation, one, I suspect, out of politeness; the remainder disliked it intensely.

Dr. Rendell states that "in about a tenth of the cases the induction, always slower than after barbiturate premedication, was delayed by vomiting the elixir." On the first point I concur wholeheartedly; the effect is most noticeable. As to the incidence of vomiting (the summary states that unpleasant side-effects were not observed), I have been more fortunate in having only two such cases, but even this incidence I regard as highly unsatisfactory when one has not oneself, nor one's colleagues, encountered a single case of vomiting during induction in many hundreds of guillotine tonsillectomies where atropine alone was the premedicant. Post-operative vomiting following methylpentynol was almost invariable.

It is asserted that the children settled quickly post-operatively, a statement at variance with the implication that "nepenthe" was needed frequently post-operatively. This bears out my own observations to the extent that ward staffs actively disliked the use of methylpentynol on account of the almost universal post-operative restlessness and lack of co-operation that accompanied its use, after which nepenthe was invariably required. This by contrast to children who, having had atropine alone pre-operatively, very rarely require any post-operative sedation at all following ethyl chloride for the guillotine operation. My clinical impression on this point is that, whereas the non-drug-sedated child is soon completely "round" after the operation and after suitable explanation and reassurance will lapse into quiet sleep,

a child who has had methylpentynol is still confused and incapable of appreciating the situation, and thus reacts accordingly. This variation between the two groups, at the same operating sessions, has been most striking, and in the latter instance has certainly increased rather than decreased the nursing care required post-operatively.

Dr. Rendell points out that in fact the ill-behaved or frightened child, whom surely it is most important to sedate, is best premedicated with a barbiturate. Despite the fact that methylpentynol was liked by ward staff and anaesthetists its use was discontinued. My own impression of methylpentynol as a sedative, which bears out Dr. Rendell's recommendation above, is that its effects are, not surprisingly, as varied and to some extent as unpredictable as the more familiar ethyl alcohol when indulged in by persons of varying temperament and mood.—I am, etc.,

Portsmouth. N. G. P. BUTLER.

SIR,—Using methylpentynol as a premedicant before tonsillectomy, Dr. Christine M. Rendell (*Journal*, December 11, p. 1397) found approximately 15% of children either tearful and apprehensive or screaming and resistant, and 85% calm and co-operative. The state of apprehension before premedication is not recorded, nor is there a comparable control series. Dr. Rendell concludes in her summary that methylpentynol "successfully diminished pre-operative apprehension." It may be argued just as reasonably, or unreasonably, that the failure rate showed pre-operative apprehension to be unaffected or increased. Indeed, the latter conclusion may be nearer the truth, as many observers will argue that these figures compare unfavourably with those following atropine premedication alone.

Dr. Rendell found that 10% of the children vomited during induction and a still greater proportion vomited in the theatre at some stage. Such vomiting may endanger life, and be distracting and disagreeable to both surgeon and anaesthetist. In the text of her article Dr. Rendell attributes this vomiting to gastric irritation induced by the drug. It is therefore a surprise to learn that the second conclusion in Dr. Rendell's summary (only too often the only part read when attempting to keep abreast of the literature) is that with this drug "unpleasant side-effects were not observed." It is unfortunate and misleading that this summary should contain such unsubstantiated conclusions divorced from Dr. Rendell's earlier statements that the drug "fell short of the ideal" and that she had since abandoned the method.

Finally, Dr. Rendell found that methylpentynol when compared with other premedicants prolonged the induction period, and that many children retained a vivid and unpleasant recollection of their experiences. Furthermore, in this series the children awaiting operation were allowed to observe the return from the theatre of their comrades, never a pleasant sight even under ideal conditions. May I be allowed to doubt yet another of Dr. Rendell's contentions, that "the importance of psychic trauma has long been realized"?—I am, etc.,

Portsmouth. R. J. HAMER HODGES.

SIR,—The old question of premedication for tonsillectomies in children is a perpetual recurrent. Atropine gr. 1/100 (0.65 mg.) by injection and a kind and calm nursing staff is the safest and most satisfactory premedication there is. After all, any premedication swallowed gives increased gastric motility and increased risk of vomiting and its sequelae, any morphine derivative depresses respiration and makes it difficult to obtain the required depth of anaesthesia, and most children object more to rectal injections than a gas tactfully given. All the barbiturates make the children restless and difficult to control post-operatively unless there is a nurse for each patient. I give anaesthetics for upwards of five ear, nose, and throat lists a week, and I remain convinced that the best and safest premedication is atropine 0.65 mg. and nothing else.—I am, etc.,

Kew, Surrey. D. B. HALSTEAD.

Appendicectomy in Chronic Appendicitis

SIR,—Professor J. Chassar Moir (*Journal*, December 11, p. 1415) has done well to draw attention to the paper by Mr. Iain McLennan and Mr. Kennedy Watt in which they claimed that the incidence of alleged chronic appendicitis is between three and four times as great in women of the menstrual age as in other females and of males of all ages.

This disparity is enough to justify strong criticism of the correctness of the diagnosis in females of the reproductive age. If it were true it would indicate a special vulnerability of the appendix as a result of the physiological processes of menstruation, but we know that the appendix and the pelvic organs have no specific association, for they belong to different parts of the body. Apart from the waste of time and energy, not to emphasize the distress caused by an unnecessary laparotomy, the patient suffers two other hazards which are even more serious. Only too often we hear after the operation that the right ovary was removed with the appendix. The general surgeon, who chiefly performs these operations, naturally cannot have the same specialized knowledge of the female pelvic organs as a gynaecologist of experience. On finding, as so frequently, a normal appendix that shows no evidence of previous inflammation, he is tempted to seek a further source of the patient's pain. The right ovary is near by and is easily picked up. It will usually contain a follicle cyst or corpus luteum, perhaps some old unruptured follicle, and is thereby incriminated and removed. The ovary seldom is the seat of pain. Perhaps in endometriosis, but not always, possibly if prolapsed with a varicocele of the pampiniform plexus of veins, and as a part of salpingo-oophoritis. It is a serious matter to remove a normal ovary from a young or not so young woman, but it is a common part of the unnecessary operation for so-called chronic appendicitis.

A second hazard is a diagnosis of "adhesions" to explain the recurrence of the old pain after a few months, which may lead to a further operation or even more than one. And thus in some introspective types of feeble personality is laid the foundation of the "chronic abdominal woman." When I was a house-surgeon one of my chiefs used to warn us "never to open the abdomen for pain alone." Unless there are some other symptoms, physical signs, a significant history, or positive tests, the surgeon will usually draw a blank. So few of these women who are diagnosed as cases of chronic appendicitis, especially the young ones, can show any supporting evidence of true appendicitis.—I am, etc.,

London, W.1. ALECK BOURNE.

SIR,—It is a fortunate thing that the lower abdominal pain and tenderness, which is so frequently associated with chronic inflammation of the cervix uteri, is rather more common on the left side than on the right. There is no doubt that this is the reason why more women than would otherwise be the case have been able to preserve their normal appendix from its surgical removal.—I am, etc.,

Glasgow, S.W.1. JAMES MAIR.

SIR,—General practitioners naturally see things differently from those whose work is limited to hospitals, and as a G.P. I am afraid that I must disagree profoundly with several of Professor J. Chassar Moir's laudable attempts (*Journal*, December 11, 1945, p. 1415) to lay down the law about chronic appendicitis. But, before I do so, may I suggest that "a more plausible explanation" is not necessarily more correct, and certainly not more scientific, especially when its plausibility lies in assuming the other fellow to be wrong. But in fact the chart of age incidence, to which he refers, has very little validity from which to argue as he does, for any general practitioner of experience knows how frequently patients deny that they have had pain or other symptoms previously until confronted with his written record of such occurrences, often years before.

From the data given the chart should be more correctly construed (with a slight shift to the right) as a chart of the time at which the patients came to operation. If this is done, several suggestions seem reasonable, the first being that the sudden increase at the age of 16 (if the relatively small numbers justify any conclusion, though they accord with my impressions) represents the time when the girls leave school and become more able to decide their destinies for themselves. Also, if the pain is at all serious it interferes with their earning capacity and makes operation more desirable. After a woman has begun to have a family she finds it increasingly difficult to get away for an operation, and all practitioners are familiar with the way women struggle on with all sorts of disabilities. Until these personal and family factors are taken into consideration it would be risky to draw even the most "plausible" conclusions from the chart, particularly as we do not know what proportion of the younger age group were treated at children's hospitals.

Professor Chassar Moir's final paragraph, "facing the facts," is quite contrary to my thirty-odd years' experience here, during which we have had the inestimable advantage of being able to operate on any case which seemed to require it. Pain may be completely absent in cases of chronic and even pretty acute appendicitis, and if present it may be felt far from the right iliac fossa or the umbilicus; there may be no apparent history of gastric disturbance. An enormous amount of time, money, and energy is consumed by "complete investigations," which are mostly unnecessary, for a patient's sensations are much more delicate than most tests, and a thorough clinical investigation on old-fashioned and, alas, outmoded lines, which paid careful attention to these, would in most cases lead to a correct diagnosis, even where the investigations are negative, as they so frequently are. Tests are most valuable in research, but in appendicitis anyhow a doctor should be above all a clinician and not a signpost to a clinical laboratory.—I am, etc.,

Winsford, Cheshire. W. N. LEAK.

Infantile Scurvy

SIR,—The reminder by Drs. G. Howells, P. E. S. Palmer, and W. H. St. John-Brooks that infantile scurvy is not extinct, and should not be forgotten in differential diagnosis, is timely (*Journal*, November 13, p. 1143). Three weeks ago a 15-month-old male infant was admitted to the Evelina Children's Hospital, under the care of Dr. B. Buckley Sharp, suffering from this disease, with a two weeks' history of painful lower limbs and a classical clinical picture. The gums were bluish, swollen, and bleeding; tender swellings were present at the lower ends of both femora and just above both ankles; and the skin was generally scaly with perifollicular hyperkeratosis on forearms and legs. There was pitting oedema of both feet and a palpable scorbutic "rosary" was present. In contradistinction to the cases quoted by the above authors, this infant was undernourished and weighed only 15 pounds (6.8 kg.). The relatively late onset was also of interest. Although the mother had had three children previously who had all maintained good health, this was the only one she had been unable to breast feed. The diet from the age of 2 months consisted of boiled cows' milk with cereals, with the addition of some orange juice diluted with *boiling* water.

The diagnosis was confirmed by the characteristic *x*-ray appearances in the long bones, a 24-hour urinary ascorbic acid excretion of 1.4 mg., and by the satisfactory response to treatment by injection of ascorbic acid 200 mg. intramuscularly on alternate days. Other biochemical features were the low levels of serum calcium (8 mg./100 ml.), and of inorganic phosphate (2.2 mg./100 ml.), and of serum alkaline phosphatase (4.5 King Armstrong units). Rickets was suggested as an additional diagnosis despite the low alkaline phosphatase. Vitamin D was also given, and these biochemical anomalies were quickly rectified (serum calcium 9.4 mg./100 ml., serum inorganic phosphate 3.4 mg./100 ml., alkaline phosphatase 26 King Armstrong units).—I am, etc.,

London, S.E.1. S. E. LEVIN.

Cantharidin Poisoning

SIR,—With reference to the last paragraph in your leading article on cantharidin poisoning (*Journal*, December 11, p. 1405), you state that Craven and Polak say "there are no grounds whatever for retaining preparations of cantharidin in the *Pharmacopoeia*." In fact, cantharidin and its preparations have not appeared in the *British Pharmacopoeia* since the edition of 1932, and you misquote Craven and Polak in having mentioned the *Pharmacopoeia* in the paper to which your leading article refers. In it they simply say that "since cantharidin preparations have no special virtue but are highly dangerous they should no longer be made available for therapeutic purposes"—a statement with which I heartily agree.—I am, etc.,

D. M. DUNLOP,
Chairman,
British Pharmacopoeia Commission.

Edinburgh.

SIR,—In the leader entitled "Cantharidin Poisoning" (*Journal*, December 11, p. 1405) a statement is made that "it (cantharidin) survives in the *British Pharmaceutical Codex* to-day in six preparations for use as counter-irritants . . ." and later that "there are no grounds whatever for retaining preparations of cantharidin in the *Pharmacopoeia*." During the recent revision of the *British Pharmaceutical Codex* it was decided that the monographs for cantharidin and its preparations should not be retained in the *Codex*, and so they have been omitted from the *B.P.C.*, 1954, which was published on October 29, 1954.—I am, etc.,

K. R. CAPPER,
Editor,
British Pharmaceutical Codex.

London, W.C.1.

Chlorpromazine

SIR,—I have been re-reading the articles and letters about chlorpromazine (*Journal*, August 21, p. 439, and September 4, pp. 560 and 565), in the light of my own, very limited, recent experience in general practice. This leads me to suppose either that my patients have been particularly unfortunate or that the sentence in your leading article (p. 581), "The drug seems to be relatively non-toxic," needs revision.

I have now given "largactil" to 18 patients, nearly all of them psychoneurotics with marked emotional tension. In no case has the dose exceeded 25 mg. three times daily or the period of administration been more than five weeks. Despite this, three patients have developed clinical jaundice. In none of these three was there any evidence in the history to suggest previous liver damage. One man of 56, who had only been taking the drug for eight days, became deeply jaundiced and remained so for over a week; he also had severe initial symptoms of vomiting, anorexia, and nausea. In the two other cases, a girl of 20 and a woman of 53, the jaundice was transient; both had been taking 25 mg. three times daily, the former for 10 days and the latter for about four weeks. In these three cases chlorpromazine therapy was stopped and has not been resumed.

One other patient had to stop taking the drug because of a severe febrile reaction after only a few days of treatment. Two others asked for therapy to be discontinued because of other side-effects. In three cases the treatment was stopped after two to three weeks because no benefit had been obtained. Five patients seem to have been helped by chlorpromazine; one of these developed jaundice and expostulated bitterly when the drug was withheld. The remaining few patients are still under treatment, the effect of which cannot yet be assessed.

In view of the anti-emetic and emotional effects of chlorpromazine, it is very tempting to use the drug in cases of hyperemesis gravidarum. In view of the danger of liver damage, I feel that this temptation should be resisted. I gave one girl with hyperemesis 40 mg. daily in divided doses. She asserted very vehemently that her vomiting was made much worse. This statement was somewhat borne out by the rapidity with which she settled down when the therapy was discontinued.

In general, my experience of chlorpromazine has been disappointing. It is not such a stand-by for the G.P. as its admittedly valuable and interesting properties would lead one to suppose.—I am, etc.,

Huddersfield. J. BRYAN EAGLES.

Flourpaper Tuberculin Jelly Test

SIR,—In the recent correspondence on tuberculin testing, it has been argued that there is some urgency to evaluate the Heaf multiple puncture test. We have been using this test in Cardiff for the past four years for child contacts, hospital staff, and, this year, as a single tuberculin test prior to giving B.C.G. vaccine to "school-leavers." When introducing our school-leaver scheme we were anxious to give B.C.G. vaccine following a single tuberculin test. For the first term we used the Heaf multiple puncture test with P.P.D. tuberculin (2 mg. per ml.) on the left arm, and the Mantoux test using P.P.D. tuberculin (10 I.T.U.) on the right arm. The results were as follows:

	Positive	Negative	Doubtful	Total
Heaf test	325	620	6	951
Mantoux	323	620	8	951

It is thus seen that in this series of children there is greater agreement between the Heaf and Mantoux tests when using P.P.D. (10 I.T.U.) than in the series reported by Dr. H. G. Calwell (*Journal*, November 27, 1954, p. 1292), when a dose of 100 units of old tuberculin was used. As Dr. Calwell has pointed out, some of the results that are regarded as positive are in fact false positives. We have retested and given B.C.G. vaccine to a number of contacts who were negative to the Heaf test but were originally regarded as positive when retested with a dose of 100 units of old tuberculin. In these contacts we have not had an accelerated or untoward reaction to the B.C.G. vaccine. As Dr. R. Stevenson Doig (*Journal*, December 4, 1954, p. 1357) points out, the advantages of the Heaf multiple puncture test are many, particularly as it can be used as a single test prior to vaccinating "school-leavers."—I am, etc.,

Cardiff. P. M. BOWEN.

Spontaneous Rupture of the Spleen

SIR,—In view of the recent discussion on the association between perforation of a peptic ulcer and rupture of the spleen, we feel that the following case throws further light on this condition.

A man aged 55 years was admitted to Poplar Hospital and operated on 10 hours after the perforation of a chronic peptic ulcer. The perforation, which was on the anterior wall of the duodenum, was closed and covered with omentum. Post-operatively the patient developed a productive cough with an evening pyrexia to 102° F. (38.9° C.), and this responded to the exhibition of sulphamethazine and streptomycin for five days. From the fifteenth day he had evening rigors for which no cause could be found. Examination of the blood showed no malarial parasites. On the twenty-first post-operative day he suddenly collapsed and died within a few minutes. Post-mortem findings: the peritoneal cavity contained three pints (1.7 litres) of blood with evidence of recent clotting. A small abscess surrounded the splenic pedicle, and there was a septic thrombophlebitis of the first inch of the splenic vein. The spleen was softened and twice the normal size, with a large subcapsular haematoma of the lower pole and an irregular tear in the capsule. The ulcer was satisfactorily sealed by omentum.

This case is similar to that recorded by Babson and Morgan.[1] Their patient, a man of 49 years, collapsed five days after an operation for the closure of a perforated duodenal ulcer. After conservative treatment, continued for six days, laparotomy was performed. The peritoneal cavity was found to contain eight litres of blood, and the spleen, with the laceration on its superior border, was removed. The patient recovered.

Mr. Blair Thompson (*Journal*, November 20, p. 1209) reported that in his case, at laparotomy, the spleen had a large subcapsular haematoma and the splenic vein contained clots. We agree with him that the progress in these cases is most likely: (1) perforation of the peptic ulcer, (2) local inflammation and thrombosis of the splenic vein, (3) congestion of the spleen leading to a subcapsular haematoma. It is difficult to imagine that severe muscular rigidity suggested by Mr. J. A. Shepherd[2] can damage an organ which is normally protected by lower ribs. The effect of coughing suggested by Babson and Morgan is similarly unlikely. The case described by Mr. Shepherd does not fall into this group: blood was not found in the peritoneal cavity until after the perforation had been discovered an inch from the oesophageal opening. It is possible that this case may come into another category—namely, traumatic rupture.

We wish to thank Mr. Alan Perry, M.S., F.R.C.S., under whose care this case was admitted, for permission to publish these details.—We are, etc.,

London, E.1. ANTHONY R. ANSCOMBE. D. J. CROWTHER.

REFERENCES

[1] *Amer. J. Surg.*, 1946, **72**, 97.
[2] *British Medical Journal*, 1949, **1**, 1082.

Chicken-neck-wringer's Finger

SIR,—The digital occupational deformities described as window-cleaner's finger by Mr. P. J. Moir (*Journal*, September 25, p. 757) and paper-folder's fingers by Dr. M. W. Partington (*Journal*, November 13, p. 1166) prompt me to record this case seen in the out-patient department at the Royal Free Hospital in 1940.

The patient was a lady aged 45 who had worked in Smithfield market from the age of 14 until she was 36 at the occupation of killing chickens. The bird was placed

across her lap, the wings and legs being held in her left hand, and, by stretching the neck with the right hand, she dislocated the cervical vertebrae with her middle finger. She stated that she could kill a bird up to the size of about 11 lb. (5 kg.) weight. She gave up the occupation when she was 36 owing to an increase in domestic duties, and the condition was quite painless until she was 45 years old, when she first noticed some pain in the joint and came to hospital.

X ray showed subluxation and osteoarthritis of the distal interphalangeal joint of the right middle finger. She was seen again a few weeks ago, at the age of 59; the condition is very little worse and gives her no trouble in her household duties.—I am, etc.,

London, W.1. GEORGE QVIST.

Professional Secrecy

SIR,—Dr. E. C. Dawson's richly informative discussion (*Journal*, December 18, 1954, p. 1474) of the doctor's duty as witness and informer may be supplemented by reference to a related problem concerning the psychiatrist. It happens, for instance, that litigants in divorce actions try to exploit the analytical treatment received by the other party, or a family member, and propose that the therapist is called as a witness and be subjected to cross-examination. It is thus hoped to elicit personal particulars damaging, or at least embarrassing, to the other party.

All persons consenting to unreserved communication in the interest of their treatment do so because they have been reassured as to the unconditional secrecy of the whole material thus disclosed. Without this absolute belief in the therapist's integrity, few people might seek this type of

treatment; and certainly very few would make satisfactory progress. Many in the legal and medical professions may not realize the widespread damage bound to result if the Press reported that a psychotherapist had been compelled to divulge anything entrusted to him by his patient, who had been made to believe that such a betrayal was impossible.

On two occasions I was approached for advice by colleagues pressed and intimidated by solicitors desirous of involving them in the lawsuit. I advised my friends to reply that the evidence thus enforced might easily result in harm to their clients' interests, and almost certainly be of no benefit to them. I have so far not heard of a case of a psychotherapist being actually forced to give evidence in such repugnant circumstances. But, if ever a psychiatrist finds himself in such a situation, ought he not to risk going to prison and cause public indignation, rather than yield to pressure and destroy the trust we psychotherapists enjoy and so much need in our professional activities? Otherwise, how could he afterwards face his current patients, or reassure new ones as to the absoluteness of professional secrecy?

We understand that ministers of religion are in practice not asked in court to divulge confessional secrets. I think this is so because the clergy would at any rate refuse to submit. This legal attitude should be extended to those handling intimate psychiatric material, even though members of the medical profession are unlikely to defend the principle in question with the same intransigence as manifested by priests. The refusal by the psychotherapist to testify would be certainly the only moral alternative where no crime or serious harm is to be prevented by divulging the patient's secrets.—I am, etc.,

London, N.W.4. S. LOWY.

SIR,—The day the Association alters its present views on professional secrecy we shall see headlined in the Press: "Doctors Can Divulge Your Secrets" (and, in smaller print) "Under Certain Circumstances." Once this has happened irreparable harm will have been done to the profession. Let us not meddle with high principles, but rather let us strive to attain them.—I am, etc.,

London, S.E.2. PETER H. SHERWOOD.

Renal Complications after Sulphonamides

SIR,—It was purely by chance that, in looking up a past reference in your *Journal*, I saw the correspondence on renal complications due to sulphonamides. Ever since seeing a case of incipient obstruction due to the use of sulphathiazole in the Tropics in 1943 a special routine was evolved, since when a case of renal complication from the use of sulphonamides (including sulphanilamide, sulphathiazole, sulphadiazine, sulphamerazine, "sulphatriad," sulphaguanidine, and sulphadimidine) in doses up to 30 gr. (2 g.) four-hourly for many days has not been encountered. Two precautions were necessary: (1) All sulphonamides except the sulphaguanidine type were given as a mixture containing as well sodium citrate 60 gr. (4 g.), the mixture being taken in a large glass of lemon squash. (2) Fluid intake *was not used as a criterion*, but each specimen of urine was kept and examined, the last specimen being thrown out of the clear glass container just before passing urine. A yellow coloration indicated not enough fluids (whatever the fluid *intake* chart said), and a very pale light green to colourless was considered just right. This was acting on the principle that it is not the fluid intake which really matters but the fluid output.

Adhering strictly to these two principles, I have not seen any case of renal complications using sulphonamides even in the Tropics, and would be very interested to hear from correspondents who have had any failures, not due to the failing of this routine itself.—I am, etc.,

Kuala Belait, Brunei State. K. F. D. SWEETMAN.

Obituary

ELIZABETH CASSON, O.B.E., M.D., D.P.M.

Dr. Elizabeth Casson, founder and medical director of Dorset House School of Occupational Therapy, Oxford, died at Bristol on December 17, 1954, at the age of 73.

Elizabeth Casson was born at Denbigh on April 14, 1881, the third daughter of Major Thomas Casson, J.P., who brought an original mind to organ building, and brother of Sir Lewis Casson, the well-known actor. After leaving St. Mary's College, Paddington, she became interested in housing estate management and worked under Miss Octavia Hill from 1908 to 1913. During this time she was secretary of Red Cross Hall at Southwark. At the age of 30 she took up the study of medicine at Bristol University, graduating M.B., Ch.B. in 1919 and proceeding M.D. in 1926. After holding a resident appointment at the West Herts Hospital in Hemel Hempstead she took the D.P.M. in 1922 and joined the staff of the Royal Holloway Sanatorium at Virginia Water. It was while she was a medical officer there that she called a meeting of medical women who were interested in psychological medicine, and from this beginning grew the Standing Committee in Psychological Medicine of the Medical Women's Federation, as well as an informal group of medical women with interests in this field who have met at intervals ever since. Dr. Casson gained the much-coveted Gaskell Prize and Medal of the Royal Medico-Psychological Association in 1927, and she later founded the Gaskell Club, the members of which are past winners of the medal. The club has met yearly for many years to welcome new medallists, to dine, and to discuss matters of common interest. Dr. Casson was always an outstanding personality at the dinners, taking the chair at the last meeting a few weeks ago.

In 1929 she returned to Bristol as medical officer in charge of a residential clinic for mental disorders, and she was also in due course appointed to the staffs of the Bristol General Hospital and the Walker Dunbar Hospital. In 1930 she founded the first school of occupational therapy in this country. It was opened at Dorset House, on the Promenade, Clifton. Dr. Casson herself wrote that the importance of occupational therapy was impressed on her when she was in hospital one Christmas and found the enormously improved atmosphere in the hospital ward was directly due to the interest of the patients in preparing the Christmas decorations. She acknowledged that her first introduction to occupational therapy came from a description by Dr. David (now Sir David) Henderson of the small department he had opened at Gartnavel Hospital, Glasgow. During the first three years of the existence of this school the training given was chiefly for mental hospital work, but in due course an occupational therapy department was opened at the Bristol General Hospital for the treat-

ment of patients with physical disease. Occupational therapists are nowadays able to take the diploma for either physical or mental work or the combined diploma for both. In the early years of the school Dr. Casson herself gave the lectures to the students on anatomy and physiology. During the war the school moved to Barnsley Hall Hospital, Bromsgrove, Worcestershire, but after the war ended it was attached to the Churchill Hospital, Headington, Oxford, where Dr. Casson was a frequent and welcome visitor. She was elected an honorary fellow of the World Federation of Occupational Therapists, and in 1951 was awarded the O.B.E. for her work as founder of the Dorset House School of occupational therapy.

In the years before the war Dr. Casson worked actively for the B.M.A. and represented her Division at nine Annual Meetings. She also took a keen interest in Bristol University and was a member of the university court. Her whole heart was in her work, and what she gained in knowledge she put to the service of others. But that was not all, for apart from her work she was affectionately devoted to innumerable friends, patients, and students and to a wide family circle. During the war her home in Bristol was bombed, and since then she had lived in Clevedon.

The death on November 19, 1954, of Dr. P. H. J. TURTON, of Heanor, Derbyshire, removed an outstanding personality from the district. He was 59 years of age. Philip Henry Job Turton was the son of the late Dr. William Henry Turton and, like his father, was a native of Heanor, where he was born on August 13, 1895. He interrupted his medical studies at Edinburgh University to serve in the R.A.M.C. in the first world war, returning to graduate M.B., Ch.B. in 1921. Four years later he proceeded to the M.D., receiving a high commendation for his thesis on the aetiology of endemic goitre, based on his own observations in Heanor. In 1929 he obtained the Manchester D.P.H. When his father, who was part-time medical officer of health for Heanor for 54 years, died in 1942 he succeeded to the appointment, which he retained until his death. For many years he was also honorary physician to Ilkeston Hospital and Heanor Memorial Hospital. He retained his particular interest in the incidence of "Derbyshire neck," and in 1933 read a paper before the Section of Epidemiology and State Medicine of the Royal Society of Medicine on the distribution of simple goitre in Derbyshire. Dr. Turton was a man of many parts who brought a keen and inquiring mind to all branches of medicine.

J. S. and T. B. F. write: It was always stimulating to attend a patient with Dr. Philip Turton. His knowledge of up-to-date research in medicine was quite striking in one who was so busy in his practice. His annual reports as medical officer of health were a delightful mixture of classical quotations and solid facts on the health of the district. He bore his recent severe illness with great fortitude and never lost interest in the people of the district, whom he served so well. To his widow and family goes our sincere sympathy in their loss.

C. M. E. writes: It was a privilege to work as Dr. Philip Turton's assistant, and, later, as his junior partner. He was outstanding as a general practitioner, being a brilliant diagnostician, and he never spared himself in the service of his patients, who thought of him not only as their doctor but as their guide, counsellor, and friend.

Dr. MARY A. T. RITCHINGS died on December 9, 1954, at her home at Swansea, where she had been in practice for over fifty years. She was 77 years of age. The daughter of a herbalist, Mary Ann Thompson Ritchings was born on July 20, 1877, and was educated privately and at Glasgow University, where she graduated M.B., Ch.B. in 1903. After graduation she held the appointment of resident medical officer at Drumcondra Hospital, Dublin, before settling in general practice at Swansea. In 1918 she was appointed M.B.E. for her work as medical officer in charge of the Y.M.C.A. auxiliary hospital at Swansea during the first world war. The first woman general practitioner at Swansea, Dr. Ritchings built up an extensive practice, mostly among her own sex, and was greatly loved and admired by her patients. Although she did not accept service under the National Insurance Act of 1911 or under the National Health Service Act of 1946, she was keenly interested in the latter during its initial stages. Of a retiring disposition and in the last few years not very active, Dr. Ritchings was not known to many members of her profession or to many of the general public, but as a young woman she was a frequent speaker on political platforms. She is survived by two sisters.—J. C. O.

Sir WILLIAM HASLETT, who died at his home at Weybridge on December 17, 1954, at the age of 88, was not only a specialist in mental and nervous disorders, but also a man who contrived to devote much time to public and political work over many years. William John Handfield Haslett was born on September 10, 1866, the son of Dr. S. Torrens Haslett, of Laghey, Co. Donegal, and was educated at the Academical Institute, Londonderry, receiving his medical training at Trinity College, Dublin, where he won first-class honours in anatomy, and at Sir Patrick Dun's Hospital, Dublin, Meath Hospital, and the London Hospital. After qualifying M.R.C.S., L.R.C.P. in 1889, he held appointments at Meath Hospital, the Poplar Hospital for Accidents, and St. Luke's Hospital for the Insane. In 1892 he became resident medical superintendent of Halliford House, a private mental hospital at Shepperton-on-Thames, in which he had a proprietorial interest, and soon afterwards began to take an active part in public affairs. This work included membership of the Staines Board of Guardians, the Middlesex Insurance Committee, and the Sunbury Urban District Council, and later he was for some time a commissioner for income tax. From 1913 to 1928 he was a member of the Middlesex County Council, and as chairman of the committee for the care of mental defectives did valuable work in connexion with the establishment of special colonies for defectives. He was also a justice of the peace. In professional circles he was a member for many years of the British Medical Association, the Royal Medico-Psychological Association, whose certificate he obtained quite early in his career, the Windsor and District Medical Society, of which he was a past president, and the Kingston Medical Society. He was chairman of the South Middlesex Division of the B.M.A. from 1914 to 1919 and represented the Division at the Annual Representative Meeting held in London in 1917. Earlier, he attended the first Annual Meeting of the Association to be held in Canada—at Montreal in 1897—and read a paper on the influence of physical upon mental disease before the Section of Psychology. In 1938 he was created a knight for his political and public services in the Spelthorne Division of Middlesex, having been for many years president of the local Conservative and Unionist Association. Just before the last war he gave up his appointment at Halliford House, but continued to render useful service as consulting physician, and some six years ago he left Shepperton to go to live at Weybridge, just across the Thames. He married Miss Norah Macan in 1922 and had one son and three daughters.

P. M. J. writes: Having just had the unusual experience of reading the obituary notices (*Journal*, December 11) of two men to whom I have been secretary, may I add a little to what has already been said of them? My outstanding recollection of Dr. N. S. GILCHRIST during the 1930's is his tremendous capacity for work—nothing stood in its way for him. He would often start out at eight in the morning in order to be with "the other side's doctor" when the latter finished his morning surgery, and he would continue driving

and examining all day and well into the evening. In those days cases in the provinces provided his recreation, because he would enjoy the change of scenery. He was a perfectionist, and had a great gift for wording his reports in such a way that the layman had no difficulty in understanding them. During the 1940 blitz it was not uncommon for him to drive through the debris and over hosepipes whilst dictating his report on the last case we had visited.

I worked for Dr. A. C. ROXBURGH only during the last three years of his life, his previous secretary having been with him for 24 years. During that time, however, I realized how much his patients loved him. His ability as a skin consultant is known by all, but only those who knew him personally appreciated that his courtesy, humour, kindness, and great humility were not a façade for his patients and the medical profession only, but they pervaded his dealings with all and sundry, even his secretary. A day or two before he died I suggested that the forthcoming publication of the tenth edition of *Common Skin Diseases* might provide something of a record. His reply was, "Oh, I don't know; let's see," and he led me to his bookcase. He always marvelled—never boasted—that his private practice kept at such a level for so long after his retirement from his hospital appointments. Indeed a great man, and a great loss to all.

Medical Notes in Parliament

Agene in Flour to be Discontinued

Mr. HEATHCOTE AMORY, Minister of Agriculture and Food, announced on December 21, in answer to a question by Mr. N. DODDS (Dartford, Lab.), that he had received a report from the subcommittee set up under the chairmanship of the Chief Medical Officer of the Ministry of Health to consider the question of flour improvers.

A detailed experimental study had been made under the aegis of the Medical Research Council, he said. The methods of flour improvement which had been examined were agene, chlorine dioxide, potassium bromate, ascorbic acid, and an aeration process. The experiments had confirmed that agene-treated flour when fed in large quantities caused fits in dogs, and had shown that none of the other improvers tested did the same. Since other methods of flour improvement existed which had not produced these symptoms in animals, the Government was of opinion that, although no ill effects in man due to the use of agenized flour had been established, effect should now be given to the decision taken in 1950 to discontinue agene. In view of this decision, the National Association of British and Irish Millers had agreed to recommend their members to discontinue the use of agene and to give an undertaking that the equipment for treating flour in this way would be removed from their mills by December 31, 1955. Some delay was inevitable, because new equipment had to be obtained to treat flour by other means. The National Association of Flour Importers had similarly agreed to recommend their members to ensure that no flour treated with agene would be imported by them. The further investigations undertaken by the Medical Research Council had revealed some differences between the effects on flour of the other methods of improvement, but the effects demonstrated were not sufficient to require the discontinuance of any of the other four improving processes. These methods of flour improvement would continue to be kept under close scrutiny, in collaboration with the milling and baking industries.

Medical Evidence on Mrs. Christofi

The HOME SECRETARY was questioned by several M.P.s on December 20 about the reasons why he had decided not to advise the Queen to exercise the prerogative of mercy and reprieve Mrs. Christofi. Sir LESLIE PLUMMER (Deptford, Lab.), who asked why he had refused to see a deputation of M.P.s, also asked if the Home Secretary would publish all the medical evidence he considered in making his decision; and Mr. S. SILVERMAN (Nelson and Colne, Lab.) asked what opportunities Dr. T. Christie, principal medical officer at Holloway Prison, had of forming an opinion about the mental condition of Mrs. Christofi, what opinion he formed, and in what circumstances other medical evidence was sought, obtained, or provided.

Major G. LLOYD-GEORGE stated in reply that Parliament had on many occasions recognized the undesirability of pressing the Home Secretary to disclose the reasons why he did or did not tender advice to the Crown with regard to the exercise of the Prerogative of Mercy in a capital case. So far as concerned the particular case of Mrs. Christofi, the principal medical officer at Holloway Prison stated in a report dated October 5 that after observation of the prisoner since July 30 he had formed the conclusion that she was insane, but was medically fit to plead and to stand trial. A copy of that report was furnished to the defence. At the trial, which began on October 25, the defence did not raise the issue of insanity.

After the dismissal of the prisoner's appeal to the Court of Criminal Appeal (he continued) it fell to him to consider the case. In view of Dr. Christie's report he appointed three distinguished and experienced doctors to conduct an inquiry in accordance with statutory provision. The three doctors reported that the prisoner was not insane, and they added that in their view she did not suffer from any minor mental abnormality which would justify them in making any recommendation on medical grounds relevant to the question of a reprieve. After very full consideration he regretfully came to the conclusion that there were no sufficient grounds for recommending any interference with the due process of law. It would be contrary to long-established practice, and open to considerable objection, to disclose any advice tendered to the Home Secretary in a case of this kind by the trial judge, or to disclose the terms of the medical report submitted by the statutory inquiry, and he was not prepared to make these documents available.

Mr. KENNETH ROBINSON (St. Pancras, North, Lab.) asked what results were obtained from electroencephalographic examination of Mrs. Christofi while she was in custody. Major LLOYD-GEORGE said that there was no such examination in this case, as the prisoner would not agree to the examination being made.

Local Medical Committees

Mr. JOHN RANKIN (Glasgow, Tradeston, Lab.) asked the Secretary of State for Scotland on December 21 who was responsible, under his regulations, for setting up the local medical committees, and what was their purpose in the administration of the general medical service. Mr. J. STUART replied that these committees were formed by the medical profession themselves, and were accorded statutory recognition by the Secretary of State. They appointed medical members to the local executive council; they must be consulted by the council on certain specified matters, and were normally consulted on other matters, affecting the general medical services; and they had a number of specific functions under the regulations, including particularly the investigation of excessive prescribing.

Mr. RANKIN said there was widespread dissatisfaction with the manner in which many of the committees were carrying out their functions, and that they were tending by their attitude to limit the doctor's right to give the type of treatment he considered necessary. As they were assuming something of a judicial attitude, should not a doctor be represented when he appeared before one of these committees? Mr. STUART said he would like to look into that. He did not wish any dictatorial attitude to be adopted by these committees. He thought they were working very well.

Mr. RANKIN said that one of the interviews to which a doctor was called started by the chairman informing him that he need not worry because on this occasion he would not be fined. Mr. STUART said he was not aware of that. If Mr. Rankin would give him details he would look into them.

Industrial Health Survey

Mr. A. BLENKINSOP (Newcastle-upon-Tyne, East, Lab.) asked the Minister of Labour on December 21 whether he was satisfied that the Advisory Committee on Industrial Health would have all the powers and resources necessary to enable them to carry out an effective survey, so as to make clear the main deficiencies of the existing health provisions in industry. Mr. HAROLD WATKINSON, the Parliamentary Secretary, stated that the committee would be an advisory body. The scheme for the development of industrial health services, including any necessary surveys, would be carried out through the Factory Inspectorate, which possessed wide powers under the Factories Acts. Mr. BLENKINSOP asked if the Minister was satisfied that they had powers to inspect conditions in some of the smaller factories, where there was most need. Mr. WATKINSON pointed out that the committee was only working at the moment on premises which came under the Factories Acts.

Mr. BLENKINSOP asked if the Minister had completed his discussions with interested bodies on the composition of the advisory committee and whether he had agreed to the inclusion of persons with experience of local government work. Mr. WATKINSON said "No." Further requests for membership of the committee had been received, and the whole subject was still under consideration. Mr. BLENKINSOP pressed the Minister to give public health authorities some place on this committee in view of their heavy responsibilities. It was fantastic, he said, not to include representation from this important section. Mr. WATKINSON replied that local authorities had only limited responsibility in industrial health. That did not prejudge the Minister's decision, which had yet to be taken.

Clinical Experiments

Mr. S. SWINGLER (Newcastle-under-Lyme, Lab.) asked the Minister of Health to what extent clinical experiments were being carried out in hospitals under the regional hospital boards; what regulations governed the conditions of such experiments; and on whose authority, and under what circumstances, clinical experiments on infants in hospital, such as that to which his attention had been drawn, were permitted to be carried out. Miss PAT HORNSBY-SMITH, the Parliamentary Secretary, replying on December 20, stated that hospital authorities were given power to conduct research by Section 16 of the National Health Service Act, 1946. Individual clinical investigations were undertaken on the authority of the clinician in charge of the case, and the Minister had not complete knowledge of the extent of all the work in progress at hospitals. Such investigations involved medical and ethical problems which were not susceptible of control by regulations.

Universities and Colleges

UNIVERSITY OF CAMBRIDGE

The Raymond Horton-Smith Prize for 1953–4 has been awarded to Dr. P. A. G. Monro.

In Congregation on November 13 the following degrees were awarded:

M.A.—W. E. Hick, M.D., Reader in Experimental Psychology; Bernard Towers, M.B., Ch.B., University Demonstrator in Anatomy; Christopher Parish, F.R.C.S., Associate Lecturer in Thoracic Surgery; L. M. Rouillard, F.R.C.S.Ed., Associate Lecturer in Plastic Surgery.

M.B., B.CHIR.—P. Gortvai.

M.B.—*E. Burkinshaw.

Dr. A. P. Waterson has been elected into a Research Fellowship at Emmanuel College.

*By proxy.

UNIVERSITY OF LONDON

The following candidates at the Institute of Psychiatry have been approved at the examination indicated:

ACADEMIC POSTGRADUATE DIPLOMA IN PSYCHOLOGY.—*Section D:* Patricia M. Bannister, H. R. Beech, Marian Black, Genevieve L. Castrodale, Esther R. Gottstein, D. Hall, J. H. G. Hewlett, V. F. W. Newham.

INFECTIOUS DISEASES AND VITAL STATISTICS

Summary for British Isles for week ending **December 11** (No. 49) and corresponding week 1953.

Figures of *cases* are for the countries shown and London administrative county. Figures of *deaths* and *births* are for the 160 great towns in England and Wales (London included), London administrative county, the 17 principal towns in Scotland, the 10 principal towns in Northern Ireland, and the 14 principal towns in Eire.

A blank space denotes disease not notifiable or no return available.

The table is based on information supplied by the Registrars-General of England and Wales, Scotland, N. Ireland, and Eire, the Ministry of Health and Local Government of N. Ireland, and the Department of Health of Eire.

CASES in Countries and London	1954					1953				
	Eng. & Wales	Lond.	Scot.	N. Ire.	Eire	Eng. & Wales	Lond.	Scot.	N. Ire.	Eire
Diphtheria	15	0	8	0		28	1	3	0	1
Dysentery	357	67	165	7		362	61	177	3	1
Encephalitis, acute	1	0		0		6	1	0	1	
Enteric fever:										
Typhoid	2	2	4	0		2	0	0	0	
Paratyphoid	7	1	0	0		9	0	0	0	
Food-poisoning	134	11		0		132	23		0	
Infective enteritis or diarrhoea under 2 years				11	16				16	19
Measles*	6,298	473	493	312	61	793	15	38	16	99
Meningococcal infection	21	0	6	0	3	36	2	9	1	
Ophthalmia neonatorum	30	6	3	0		25	2	5	0	
Pneumonia†	768	49	212	4	3	583	41	183	8	4
Poliomyelitis, acute:										
Paralytic	30	2	11	1		54	3	17	2	2
Non-paralytic	13	2				34	4			
Puerperal fever§	214	33	6	1		223	38	13	3	2
Scarlet fever	709	39	151	66	37	1,613	85	268	46	74
Tuberculosis:										
Respiratory	723	85	134	51				123	23	
Non-respiratory	85	13	22	11				22	3	
Whooping-cough	2,047	123	165	25	56	2,510	106	318	68	81

DEATHS in Great Towns	1954					1953				
	Eng. & Wales	Lond.	Scot.	N. Ire.	Eire	Eng. & Wales	Lond.	Scot.	N. Ire.	Eire
Diphtheria	1	0	0	0	0	0	0	0	0	0
Dysentery	1	0		0		0	0	1	0	
Encephalitis, acute		0			0		1			0
Enteric fever	0	0	0	0		0	0	0	0	
Infective enteritis or diarrhoea under 2 years	5	0	0	1	1	6	1	3	1	0
Influenza	56	5	1	0	0	11	1	1	0	1
Measles		0	0	0	0		0	0	0	0
Meningococcal infection		0	0				1	2		
Pneumonia	301	43	19	8	12	237	31	28	9	5
Poliomyelitis, acute	2	1			0	3	0			0
Scarlet fever		0	0	0	0		0	0	0	0
Tuberculosis:										
Respiratory	112	18	18	1	3	87	10	16	1	6
Non-respiratory		0	20	0	1		0	1	2	1
Whooping-cough	0	0	1	0	0	3	0	1	0	0
Deaths 0–1 year	226	32	23	6	12	215	28	29	9	25
Deaths (excluding stillbirths)	5,703	762	589	110	189	5,165	728	561	88	187
LIVE BIRTHS	6,646	999	798	200	311	6,918	1036	814	210	321
STILLBIRTHS	186	25	24			182	21	18		

* Measles not notifiable in Scotland, whence returns are approximate.
† Includes primary and influenzal pneumonia.
§ Includes puerperal pyrexia.

Vital Statistics

Scottish Life Tables

The remarkable decline in mortality rates of young people during the last eight decades in Britain is reflected clearly in the life tables for Scotland based on the 1951 census.

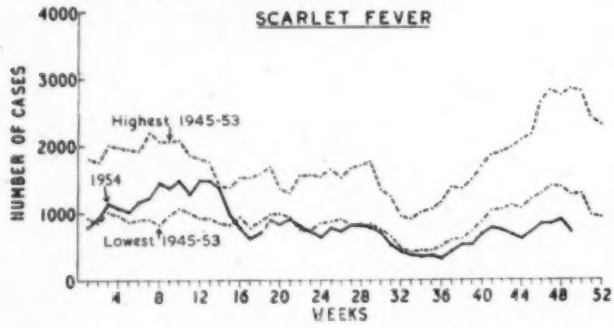
The expectation of life at birth in 1871 was 39.8 years for males and 42.0 for females; in 1951 it was 64.4 for males and 68.7 for females. But for those people who reached the age of 60 the increase was much less striking—from 13.3 to 14.3 years for males and from 14.8 to 16.8 years for females.

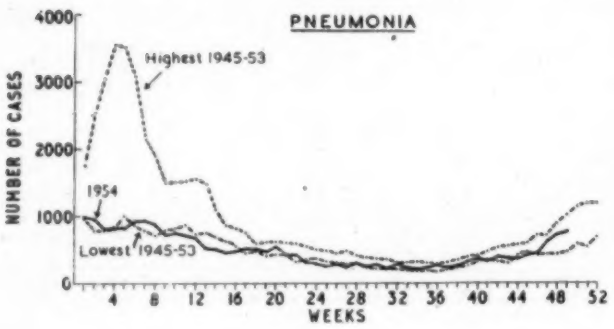
Middle childhood is the age at which mortality has been most sharply reduced. At age 10 mortality in 1951 was only 9% for males and 6% for females of mortality in 1871. At age 60 it was 79% for males and 58% for females of mortality in 1871, while at age 80 mortality was slightly worse in 1951 for males, being 105% of the 1871 rate and 100% for females.

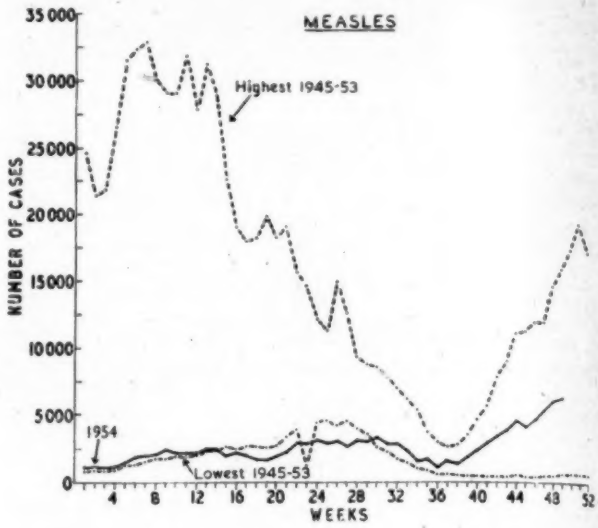
The mortality of married persons is lighter than that of single or widowed for men in all age groups, and lighter for younger women; the mortality of older women is much the same in all three categories. In the twenty years between 1930–2 and 1950–2 a change has been noted in the mortality rates of single and married women. In 1930–2 the single women's mortality rates were less than those of married women; in 1950–2 they were some 60% greater on average. Two probable reasons are advanced to account for this. First, the risks of childbirth were greatly reduced; secondly, marriage rates were high, with the result that those who remained unmarried included an unusually high proportion of women who were unmarried because of ill-health. (*Life Tables 1950–2*. Supplement to the 99th Annual Report of the Registrar-General for Scotland. H.M.S.O. 1s. 3d.)

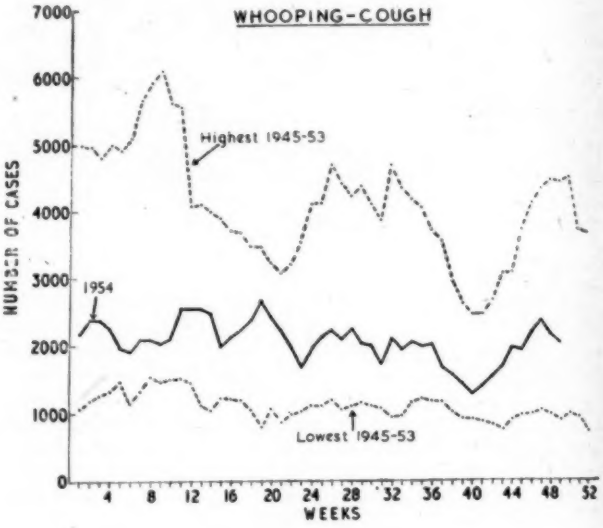
Graphs of Infectious Diseases

The graphs below show the uncorrected numbers of cases of certain diseases notified weekly in England and Wales. Highest and lowest figures reported in each week during the nine years 1945–53 are shown thus - - - - - -, the figures for 1954 thus ————. Except for the curves showing notifications in 1954, the graphs were prepared at the Department of Medical Statistics and Epidemiology, London School of Hygiene and Tropical Medicine.









Infectious Diseases

In England and Wales during the week ending December 11 a fall was recorded in the number of notifications for scarlet fever of 184, from 893 to 709, 109 for whooping-cough, from 2,156 to 2,047, and 106 for dysentery, from 463 to 357, and a rise of 281 was recorded for measles, from 6,017 to 6,298.

The largest increases in the incidence of measles were 137 in Cheshire, from 304 to 441, 83 in London, from 390 to 473, 72 in Yorkshire West Riding, from 408 to 480, and 63 in Staffordshire, from 162 to 225, and the largest fall was 71 in Nottinghamshire, from 207 to 136. The largest falls in the number of notifications of scarlet fever were 32 in Yorkshire West Riding, from 100 to 68, 30 in London, from 69 to 39, and 29 in Lancashire, from 132 to 103. The largest variations in the returns of whooping-cough were a rise of 47 in Kent, from 114 to 161, and a fall of 53 in Lancashire, from 271 to 218. 15 cases of diphtheria were notified, being 2 more than in the preceding week. The largest returns of diphtheria were Liverpool C.B. 5, and Birmingham C.B. 3.

43 cases of acute poliomyelitis were notified during the week. These were 1 more for paralytic and the same for non-paralytic cases as in the preceding week. The largest returns were Yorkshire West Riding 5 (Leeds C.B. 3), Lancashire 5 (Manchester C.B. 3), Cheshire 4 (Wallasey C.B. 2), and London 4.

The number of notifications of dysentery was the lowest for the past ten weeks. The largest centres of infection were Lancashire 96 (Oldham C.B. 24, Liverpool C.B. 21,

Morecambe and Heysham M.B. 11), London 67 (Hackney 25, Islington 10), Yorkshire West Riding 37 (Sheffield C.B. 25), Staffordshire 27 (Stoke-on-Trent C.B. 17), and Norfolk 22 (Norwich C.B. 17).

56 deaths were attributed to influenza in the great towns. The number of deaths is at a higher level than in recent years; in the past month there have been 156 deaths, compared with 56 in 1953 and 65 in 1952 during the corresponding period.

Week Ending December 18, 1954

The notifications of infectious diseases in England and Wales during the week included: scarlet fever 798, whooping-cough 1,833, diphtheria 18, measles 6,523, acute pneumonia 736, meningococcal infection 24, acute poliomyelitis 36, dysentery 340, paratyphoid fever 9, typhoid fever 1.

Medical News

Royal Society.—Once again the medical profession is honoured by the fact that both the president of the Royal Society and one of the four vice-presidents are medical men. Dr. E. D. ADRIAN, O.M., was re-elected president at the annual meeting in December, and among the members of the council are Sir EDWARD MELLANBY, a vice-president, Sir ALAN DRURY, and Professor J. H. GADDUM. The Royal Society has awarded Locke Research Fellowships to Dr. M. DE BURGH DALY, lecturer in physiology at University College, London, to support work on the haemodynamics of the pulmonary circulation, and to Dr. J. C. F. POOLE for work at the Sir William Dunn School of Pathology, Oxford, on the effects of fat absorption on blood coagulation and thrombosis.

Psychoanalysis at McGill University.—With the appointment of Dr. W. CLIFFORD SCOTT as associate professor in the department of psychiatry, a full psychoanalytical training programme will now be offered at McGill University, Montreal, for the first time. Dr. Scott graduated at Toronto in 1937, and then studied under the late Professor ADOLF MEYER and Professor MACKFIE CAMPBELL. Until recently Dr. Scott was director of the London Clinic of Psychoanalysis and chairman of the Board of Directors of the Institute of Psychoanalysis. He was also on the staff of the Bethlem Royal and Maudsley Hospital. Applications for psychoanalytical training should be made directly to Dr. SCOTT, Allan Memorial Institute, McGill University, Montreal.

Impressions of the Russian Doctors.—Before they left on December 23 at the end of their three-week tour the group of Soviet doctors who have been visiting Britain gave their impressions of medicine here (see *Journal*, December 11, p. 1431, and December 18, p. 1495). Professor A. N. SHABANOV, who led the delegation, said that they were leaving England with a good deal of satisfaction at all the things which they had seen, and about which they would tell their colleagues on their return. This exchange of scientific information was of great importance, because it gave a true understanding of the work being carried out in each country. He hoped that there would be exchange of technical information in the future, and that the contacts which the delegation had established in this country would lead to further invitations to Soviet scientists to come to Britain. Professor Shabanov then announced that he had invited British surgeons to a conference of surgeons to be held in Moscow next month, of which he was a member of the organizing committee. The Soviet doctors, Professor Shabanov continued, left with a good impression of all the medical institutions which they had visited. One specific feature of medicine characteristic of this country was the system of general practitioners, which seemed popular. It was at first very difficult for the visitors to understand the National Health Service. They had got to know quite well that part which was administered by local government, but they had seen less of the part administered by the Ministry of Health. Britain had not such a unified health service as in the U.S.S.R., where all institutions were under the Health Ministry. All the doctors whom they saw were carrying out their work with a sense of responsibility. In the medical centres and institutions which they had visited the work being done was of a high standard. A woman member of the delegation, Dr. Z. M. DZHAMALOVA, deputy Minister of Health, Uzbek Republic, an obstetrician and gynaecologist, said that the antenatal clinics were well organized in Britain and the obstetrical departments were very good. Methods of anaesthesia in childbirth differed from those in the Soviet Union, where pain in childbirth was alleviated by psychological preparation and drugs and a short application of ether in the second stage where medically indicated.

Hostel for Deaf Children.—The Astor Shield, presented by Lord Astor for outstanding work in the field of infant welfare, has been awarded this year to the Royal National Throat, Nose, and Ear Hospital in recognition of its work in opening a hostel for deaf babies and their mothers. Dr. D. H. GEFFEN, vice-chairman of the National Baby Week Council, made the presentation at a small function at the hospital on December 15. The hostel is at Ealing and provides for the accommodation of mothers who can stay with their deaf babies and very young children in order to learn to apply a deaf aid to a child at a time when there is a readiness to listen and later to talk. Dr. Geffen described this as the finest concept in child welfare that his council had come across during the last few years. If young children were to learn to talk it must be through the mother, and this was the only way of really doing something for a sadly neglected portion of the population—namely, congenitally deaf children. Miss EDITH WHETNALL, director of the audiology unit of the hospital, whose lecture on the management of deaf children was reported in our last issue (December 25, p. 1542), said that mothers and babies were kept at the hostel for from three weeks to a month. The house had been bought by the hospital, but the Nuffield Foundation was responsible for the salary of the teacher, the mothers receiving a grant from the Ministry of Health, if needed, to enable them to take advantage of its facilities.

Anatomical Society of Great Britain and Ireland.—At the annual meeting at Guy's Hospital Medical School on November 26 Professor W. J. HAMILTON and Professor F. GOLDBY were re-elected president and secretary respectively. Professor E. W. WALLS was elected treasurer in place of Professor T. NICOL.

Wright-Fleming Institute of Microbiology.—Professor ROBERT CRUICKSHANK has been appointed Principal of the Wright-Fleming Institute of Microbiology in succession to Sir ALEXANDER FLEMING, who retired on December 31, 1954. Professor Robert Cruickshank will continue to hold the chair of bacteriology in St. Mary's Hospital Medical School.

Wellcome Historical Medical Museum.—For the past seven years the Wellcome Historical Medical Museum in London has been carrying on its activities at 28, Portman Square. It has now removed to the Wellcome Research Institution at 183–193, Euston Road, London, N.W.1 (Tel. Euston 4688). Exhibitions dealing with primitive medicine and with child welfare through the ages are open to the public in the new premises (Monday to Friday, 10 a.m. to 5 p.m.).

News in Brief.—Sir LIONEL WHITBY will be visiting professor of medicine at the New York State University College of Medicine this month.

Oxytetracycline ("terramycin"), and preparations of it, intended for parenteral injection, have been brought under the Therapeutic Substances Act, 1925, by Regulations

coming into force on January 1. The provisions relating to insulin under the Therapeutic Substances Regulations, 1952, have also been amended.

At the Swedish National Medical Congress in Stockholm last month a new Swedish vaccine against poliomyelitis was announced. It is reported to be less likely to give rise to allergic reactions than the corresponding American product.

The inaugural meeting of the Polarographic Society was held in London recently. The society is open to all interested in polarography. The secretary is Mrs. B. LAMB, 25, Grittleton Avenue, Wembley.

COMING EVENTS

Institute of Diseases of the Chest.—Open clinical demonstrations on various aspects of chest disease are given on Fridays at 5 p.m. in the lecture room of the Institute (in the grounds of the Brompton Hospital, Fulham Road, London, S.W.3). These demonstrations are intended for candidates for higher qualifications. They are open without fee to medical practitioners on signing the attendance book. The next series will begin on January 7 and end on April 1.

London County Medical Society.—Annual dinner on January 18 at the Talbot Restaurant, 64, London Wall, London, E.C.2, at 7 for 7.30 p.m. Dr. ROBERT FORBES, secretary of the Medical Defence Union, will speak on "Recent Developments in Medical Litigation." Tickets 11s. 6d., from Dr. A. M. RAMSAY, Royal Free Hospital, Lawn Road, London, N.W.3.

Israeli Scientific Publications.—The exhibition at the Consulate of Israel, 18, Manchester Square, London, W.1, will remain open until January 7.

Goulstonian Lectures.—Dr. G. M. BULL will lecture at the Royal College of Physicians, Pall Mall East, at 5 p.m. on January 18 and 20 on "The Uraemias."

Pan American Congress of Ophthalmology.—Fifth congress in Santiago, Chile, January 15–22, 1956. Details from Dr. RENE CONTARDO, Huerfanos 930—Of. 74, Santiago, Chile.

Association of Industrial Medical Officers.—The association will be celebrating the 21st anniversary of its foundation by holding a special anniversary meeting in London from September 25 to 29, 1956.

NEW ISSUES

Archives of Disease in Childhood.—The next issue (Vol. 29, No. 148) is now available. The contents include:

IDIOPATHIC HYPERCALCAEMIA IN AN INFANT. I. M. P. Dawson, W. S. Craig, and J. J. C. Perera.
THE INVESTIGATION OF A LARGE FAMILY AFFECTED WITH VON WILLEBRAND'S DISEASE. J. C. W. MacFarlane and M. J. Simpkiss.
NORMAL RENAL FUNCTION IN THE FIRST TWO DAYS OF LIFE. R. A. McCance and E. M. Widdowson.
THE INFLUENCE OF EVENTS DURING THE LAST FEW DAYS IN UTERO ON TISSUE DESTRUCTION AND RENAL FUNCTION IN THE FIRST TWO DAYS OF INDEPENDENT LIFE. R. A. McCance and E. M. Widdowson.
PROGNOSIS IN IDIOPATHIC THROMBOCYTOPENIC PURPURA OF CHILDHOOD. G. M. Komrower and G. H. Watson.
CHRONIC GENERALIZED OEDEMA AND HYPOPROTEINAEMIA OF OBSCURE ORIGIN. R. S. Illingworth and E. Finch.
TEMPORARY GENERALIZED OEDEMA OF OBSCURE ORIGIN. R. S. Illingworth and E. Finch.
COLLODION SKIN OF THE NEWBORN. O. D. Fisher and Kathleen G. McKee.
CONTROL OF THE BLOOD SUGAR LEVEL IN THE NEONATAL PERIOD. James W. Farquhar.
FIVE CASES OF ACUTE INFECTIVE POLYNEURITIS (GUILLAIN-BARRÉ SYNDROME) IN CHILDREN. Pamela Aylett.
THE TREATMENT OF TICS IN CHILDHOOD. David M. Zausmer.
THE RETINOPATHY OF PREMATURITY. J. D Kerr and G. I. Scott.
A CONTROLLED INVESTIGATION ON THE EFFECT OF DIET ON ACUTE NEPHRITIS. R. S. Illingworth, M. G. Philpott, and John Rendle-Short.
TRICHOBEZOAR. F. C. Friedlander and P. Kushlick.
RADIOIODINE (I^{131}) IN JUVENILE MYXOEDEMA DUE TO ECTOPIC THYROID TISSUE. E. M. McGirr and James H. Hutchison.
NEONATAL ASCITES ASSOCIATED WITH URETHRAL OBSTRUCTION. N. E. France and E. H. Back.
CHRONIC MYELOGENOUS LEUKAEMIA IN INFANCY. Edward G. Fox and Janet L. P. Hunter.
NON-ENDEMIC GOITROUS CRETINISM. A. D. M. Jackson.
THE TREATMENT OF HYPOPLASTIC ANAEMIA AFTER CHLORAMPHENICOL. R. McLaren Todd.

Issued six times a year; annual subscription £3 3s.; single copy 12s. 6d.; obtainable from the Publishing Manager, B.M.A. House, Tavistock Square, London, W.C.1.

Medical and Biological Illustration.—The new issue (Vol. IV, No. 4) is now available. The contents include:

SOME CONDITIONS OF THE NAILS. Part of a Teaching Display from Westminster Hospital Medical School.
JOSEPH LEIDY. W. J. Bishop.
THE VALUE OF GRAPHICAL MATERIAL. M. D. Vernon.
MOTION STUDY. John Hall
THE PHOTOGRAPHY OF PAPER CHROMATOGRAMS. J. P. Berry, J. D. Cheshire, and L. I. Woolf.
SELF-CONTAINED UNIT FOR KINEMICROGRAPHY WITH BUILT-IN EXPOSURE DEVICE. H. M. Dekking.
EQUIPMENT AND METHODS:
THE "MICROMAG" FOCUSING DEVICE.
THE LEDA NUMBERING MACHINE.
EQUIVALENT FOCAL LENGTHS.
THE BRUBACKER-HOLINGER 16 MM. ENDOSCOPIC MOTION PICTURE CAMERA.
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SOCIETIES AND LECTURES

A fee is charged or a ticket is required for attending lectures marked ●. Application should be made first to the institution concerned.

Monday, January 3

INSTITUTE OF DERMATOLOGY.—Fourth of a series of semi-permanent exhibitions from January 3 to 21, by Dr. J. A. Milne, on Histology of Acute Vascular Disease of the Skin.
INSTITUTE OF NEUROLOGY.—5 p.m., Sir Geoffrey Jefferson, F.R.S.: Benign and Malignant Tumours of the Gasserian Ganglion.

Tuesday, January 4

INSTITUTE OF DERMATOLOGY.—5.30 p.m., Dr. L. Forman: Skin Lesions with Deeper Significance.
ROYAL COLLEGE OF SURGEONS OF ENGLAND.—3.45 p.m., Erasmus Wilson Demonstration by Dr. G. D. Lumb: Some Pathological Aspects of Ulcerative Colitis.

Wednesday, January 5

INSTITUTE OF DERMATOLOGY.—5.30 p.m., Dr. H. Haber: Parapsoriasis, Poikiloderma, and Mycosis Fungoides.
INSTITUTE OF LARYNGOLOGY AND OTOLOGY.—5.30 p.m., Professor F. C. Ormerod: Indications for the Use of Antibiotics in Otolaryngology.

Thursday, January 6

ROYAL ARMY MEDICAL COLLEGE.—5 p.m., Centenary Lecture by Dr. Muriel Robertson, F.R.S.: Sir David Bruce—An Appreciation of the Man and His Work (lantern slides).
ROYAL COLLEGE OF SURGEONS OF ENGLAND.—5 p.m., Hunterian Lecture by Mr. J. M. Potter: Angiomatous Malformations of the Brain: Their Nature and Prognosis.
ST. JOHN'S HOSPITAL DERMATOLOGICAL SOCIETY.—4.30 p.m., demonstration and discussion of clinical cases.

Friday, January 7

●INSTITUTE OF DERMATOLOGY.—5.30 p.m., clinical demonstration by Dr. J. E. M. Wigley: Psoriasis.
INSTITUTE OF DISEASES OF THE CHEST.—5 p.m., clinical demonstration by Dr. J. Smart: Congenital Abnormalities of the Chest.
●ROYAL FACULTY OF PHYSICIANS AND SURGEONS OF GLASGOW.—5 p.m., Dr. I. Anderson: Electrolyte Balance.
WHIPPS CROSS HOSPITAL MEDICAL SOCIETY.—8.30 p.m., clinical meeting. Members of the Stratford Division, B.M.A., are invited.

Saturday, January 8

MIDLAND TUBERCULOSIS SOCIETY.—At Birmingham Chest Clinic, 3 p.m., Dr. J. Morrison Smith: Polythene Plombage; Dr. P. A. Thorn: Gastrectomy and Tuberculosis; Dr. R. G. May: Some Observations on Segmental and Lobar Opacities in Primary Tuberculosis in Children.

BIRTHS, MARRIAGES, AND DEATHS

BIRTHS

Blend.—On December 18, 1954, at the London Hospital, London E., to Martha, wife of Dr. David Blend, 22, Compton Terrace, London, N., a son—Jonathan William Eliot.

DEATHS

Bryce.—On December 13, 1954, at 2, Kennedy Gardens, St. Andrews, Fife, William Henderson Bryce, M.B., C.M.
Cameron.—On December 12, 1954, at Kingston, Jamaica, John Joseph Cameron, M.D., aged 72.
Eunson.—On December 3, 1954, Leonard Harvey Eunson, M.C., M.B., Ch.B., of Dudley Port House, Tipton, Staffs, aged 58.
Goodwin.—On December 15, 1954, Edward Knox Goodwin, M.R.C.S., L.R.C.P., of 2, Marten Road, Folkestone, Kent, aged 86.
Iles.—On December 12, 1954, at Starcross, Devon, John Hyde Iles, M.B., B.Ch., aged 76.
Saaler.—On December 9, 1954, at Golders Green, London, N.W., Bruno Carl Saaler, M.D., aged 71.
van Someren.—On December 4, 1954, in hospital at Newcastle, Staffs, Gerald Antony van Someren, M.R.C.S., L.R.C.P., D.P.M., of Mount St. George, Second Avenue, Porthill, Stoke-on-Trent, Staffs, aged 43
Wallace.—On December 13, 1954, at Simpsons, Glenalmond, Perth, Alexander Frank Wallace, M.C., M.D., formerly of Northern Rhodesia.
Watson.—On December 13, 1954, at an infirmary, Glasgow, Henry Currie Watson, M.C., M.B., Ch.B., of 187, Neilston Road, Paisley, Renfrewshire.

Any Questions ?

Correspondents should give their names and addresses (not for publication) and include all relevant details in their questions, which should be typed. We publish here a selection of those questions and answers which seem to be of general interest.

Risk of Agranulocytosis with Sulphonamides

Q.—*What is the incidence of agranulocytosis with the commoner sulphonamide preparations? Are any more dangerous than others in this respect, and is there a maximum dosage which if not exceeded is likely to prevent the occurrence of this complication?*

A.—It is not possible to give a precise figure for the incidence of agranulocytosis during sulphonamide therapy, since many cases are either not recognized or are not reported. It is probably of the order of one in a thousand to one in ten thousand of cases treated. There is no convincing evidence that one sulphonamide is more likely to produce agranulocytosis than another, but sulphadimidine and sulphadiazine are probably safer in this respect than sulphathiazole and sulphapyridine. Most of the reported cases have arisen after heavy dosage for a week or more, or after the prolonged use of small doses as in the prophylaxis of acute rheumatism. As a crude guide it may be said that the total dose should not exceed 50 grammes nor the period of administration twenty-one days. If these figures are exceeded the patient should have weekly total and differential white cell counts carried out. Administration of a sulphonamide should be immediately stopped in any patient showing drug fever or developing a sore throat, and the blood picture should be investigated.

Treatment of Agranulocytosis following Drugs

Q.—*What is the best treatment for agranulocytosis following drug therapy? How may the prognosis be assessed?*

A.—Many substances, including pentose nucleotide, pyridoxine, folic acid, liver extracts, and A.C.T.H. and cortisone, have been used in the treatment of the agranulocytosis following drug therapy. However, there is no convincing evidence that any of these remedies has a specific effect in stimulating leucopoiesis under these circumstances. The transfusion of leucocytes is likewise of little or no value. Fortunately, as for instance in thiouracil agranulocytosis, the dangerous phase of acute granulocytopenia usually lasts only for a few days and seldom persists longer than one week. After stopping the responsible drug at once, all that is necessary, therefore, is for the patient to be kept under observation in bed so that any septic lesions which develop may be vigorously treated by an appropriate antibiotic.

The prognosis can be assessed by marrow puncture. This may show signs of leucocyte regeneration a day or two before the neutrophil count in the peripheral blood starts to rise. If the marrow puncture also reveals depression of erythropoiesis and thrombopoiesis, as in sensitivity to chloramphenicol, the outlook is much more serious than when leucopoiesis alone is depressed.

Mechanism of Blood Clotting

Q.—*What is the modern view of the mechanism of blood coagulation?*

A.—The following is an outline of the modern theory of blood coagulation. Blood clots when the protein fibrinogen is changed to fibrin. This change is brought about enzymatically by thrombin. Thrombin does not exist normally in blood *in vivo*—if it did the blood would clot in the blood vessels; it is formed from a precursor, prothrombin. It is the processes which result in the activation of prothrombin to thrombin that have been the subject of the greatest controversy and research in recent years.

It is now believed that the activation of prothrombin is brought about by "intrinsic" thromboplastin, and that this thromboplastin is formed as the result of the interaction of at least five factors in the presence of ionized calcium. Four of the five factors exist normally in the plasma—antihaemophilic globulin, the Christmas factor, and Factors V and VII; the fifth factor is derived from blood platelets as the result of contact with a water-wettable surface.

Strongyloidiasis

Q.—*I have a male patient who became infected with Strongyloides stercoralis while in a Japanese P.O.W. camp. This condition persists in spite of intensive treatment with gentian violet, and he has frequent attacks of urticaria and pruritus. What further measures can be taken? Would phenothiazine be of any value?*

A.—It is now generally agreed that there is no effective treatment for infection with *Strongyloides stercoralis*. There is no evidence that phenothiazine is of any value in such infections. Fortunately, these worms very seldom give rise to symptoms, and patients harbouring them may generally be reassured and encouraged to ignore the fact that they have a worm infection. In the case in question it is possible that the urticaria is the result of sensitivity to the worms and their products, but other causes of urticaria should be excluded. If it is decided that the *Strongyloides stercoralis* are responsible for the urticaria, symptomatic treatment should be employed.

Skin Ink

Q.—*Can you give me the prescription for an efficient skin dye for the pre-operative marking of varicose veins? Those that I have already tried are immediately removed by the application of surgical spirit.*

A.—An efficient skin ink for the pre-operative marking of varicose veins is a solution made up as follows:

℞	Pyrogallol	1 g.
	Methylated spirit	20 ml.
	Liquor ferri perchlor. ..	2 ml.
	Acetone	10 ml.
	Hydrochloric acid	1 drop

After the patient has been shaved and bathed, he should stand in a good light while the veins are marked by a pledget of cotton-wool dipped in the ink and held in dissecting forceps. The longer the interval between the application of the skin ink and the pre-operative skin preparation the more indelible the ink becomes. An interval of 12–18 hours is advisable, and even then spirit must not be scrubbed over the markings or some will be removed. The dye gradually wears off during the usual post-operative desquamation once baths are resumed.

Scarlet Fever and Nephritis

Q.—*Is it correct to treat scarlet fever cases, however mild, by rest in bed for at least two weeks in order to reduce the incidence of nephritis? If this is so, should not all cases of haemolytic streptococcal sore throat be treated in the same way for the same reason?*

A.—Post-scarlatinal nephritis is regarded, probably correctly, as a toxi-allergic manifestation. There is no conclusive evidence that patients allowed up early are in greater danger of developing nephritis. When the disease was common and dangerous, nephritis occurred even in the most carefully nursed cases, but only rarely in those efficiently treated with antitoxin. Nevertheless, it is probably unwise to submit to strain and overactivity organs that have recently

been subjected to a toxic or bacterial attack, and for this reason alone it is wiser to allow reasonable rest after the temperature has subsided.

Many authorities allow the discharge of patients from hospital about one week after onset of the disease; it is commonplace to release patients in from 10 to 12 days. And this would not be practised if it were thought that a greater risk of nephritis would result.

Not all streptococcal throats are caused by scarlatinal strains, and some scarlatinal strains are poor toxin producers. The nephritic reaction is almost certainly linked with the specific toxin. Furthermore, the patient with a scarlatinal streptococcal tonsillitis may have a high immunity to the toxin. It is probable, therefore, that streptococcal throats without rash are even less likely than scarlet fever to end in nephritis.

This answer is taken from the third book of *Any Questions?* Copies of both the second and third books of *Any Questions?*, each containing some 200 expert answers, are available from the Publishing Manager, B.M.A. House, Tavistock Square, London, W.C.1, or through any bookseller, price 7s. 6d. a copy (postage 6d.). The third book contains a cumulative index.

Virtues of Hair-oil

Q.—*In India hair-oil is used practically by everyone, partly as a fashion and partly owing to the popular belief that oil is necessary to protect the hair. It is also believed that oil protects the head against the heat and thus helps to prevent sunstroke. During my stay in England I saw that hair-oil was used very little. Is there any truth in the fact that oil is beneficial to the hair, particularly in the Tropics?*

A.—It would seem that the use of hair-oil to-day by Indians, as with many other races, is essentially for cosmetic reasons and is practised as a toilet routine with little other purpose.

There is a natural tendency to maintain a definite "head" of sebaceous secretion in the skin, and this is soon restored if grease is removed from the skin, so that the addition of more oil would not seem to have any physiological value. Climatic conditions, particularly heat, determine the normal secretion of sebum, and the natural output of the skin and scalp is no doubt adequate for all protective purposes. The coloured races secrete much larger quantities of sebum in their native climate when in good health. Ill-health is commonly indicated by a loss of greasiness of the skin, and this may have led to the idea that the addition of oil, even in health, is of value.

Furthermore, from the earliest times there has been an association between the anointing with oil and the driving out of evil influences, the conferring of divine grace and blessing, and a general sense of protection. This ritual may perhaps underlie the widespread use of oil on the hair. It is doubtful if there is any physical benefit resulting from the practice.

Hypercholesterolaemia

Q.—*What are the present views on the aetiology of hypercholesterolaemia, its relationship to coronary thrombosis, and treatment by dieting and the administration of oestrogens?*

A.—The cholesterol level in the blood depends upon the amount eaten in the diet and formed in the body on the one hand, and upon the amount excreted in the faeces and metabolized on the other. Although in animals overfeeding with cholesterol produces atheroma, it is now thought that in man the amount of ingested preformed cholesterol is less important in influencing the blood level than the amount being synthesized and metabolized in the body.

There is an association between premature arteriosclerosis and diseases characterized by hypercholesterolaemia such as occurs as a familial disorder of lipid metabolism in familial xanthomatosis, in nephrosis, and in diabetes. There is little doubt that coronary thrombosis relatively early in life can be correlated in many instances with abnormally high and fluctuating serum-cholesterol levels.

In some subjects, but not in all, a cholesterol-free diet may lower the plasma cholesterol level, and there is evidence that in women the cholesterol concentration fluctuates throughout the menstrual cycle, the lowest levels being found at the time of maximum oestrogen secretion. Whether a cholesterol-free diet or oestrogens will prove of value in preventing atherosclerosis by lowering the plasma cholesterol level remains to be proved.

Corns Between the Toes

Q.—*I should be grateful for advice on the treatment and prevention of painful soft corns between the toes in an elderly woman.*

A.—In very many cases soft corns between the toes are the result of fungus infection. A careful search should therefore be made for the organism and if it is found appropriate treatment instituted. Failing this, treatment should aim at keeping the interdigital folds slightly separated and dry. The skin between the toes should be washed night and morning with soap and water, swabbed with surgical spirit, and freely dusted with talcum powder. A few thicknesses of gauze will keep the opposing surfaces apart.

A Case for Thoracoplasty

Q.—*A man of 48 years suffered from tuberculosis of the left apex with cavitation which has responded satisfactorily to rest and chemotherapy, but for which it is considered that a thoracoplasty should now be made. The left lower lobe, however, invariably produces showers of persistent unchanging fine crepitations. On radiography, screening, bronchography, and bronchoscopy no abnormality is detected. The right lung is apparently normal. The patient has no sputum. Vigorous physiotherapy has not altered the signs. What is the nature of the disturbance in the left lower lobe and is it safe to perform thoracoplasty on that side?*

A.—A shoal of fine crepitations is sometimes an early indication of a spread of disease which has not yet become apparent radiologically. Occasionally it indicates a minor degree of fibrosis. Before considering operation it might be advisable to wait for several weeks to see if there are any radiological changes. On the assumption that there is no change it should be possible to carry out a thoracoplasty in slow stages (2 to 2½ ribs at a session). The main risk would be basal atelectasis, but if bronchostenosis has been excluded by bronchoscopy and bronchograms this should not be allowed to weigh unduly. Antibiotics should be used before and during surgery.

NOTES AND COMMENTS

Health Education.—Dr. JOHN BURTON, medical director of the Central Council for Health Education, writes: In contrast with the Hospital Service Group at the Central Council for Health Education Summer School for 1954 (see *Journal*, November 27, p. 1263), groups concerned with public health accepted in principle the need for a trained health educator. In one case it was suggested that this officer should be on the staff of the M.O.H. and responsible to the latter.

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